Essentials of

ORTHOPEDICS

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Second Edition

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Foreword
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Essentials of Orthopedics

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Dedicated to

My Wife Renu
and
Daughter Archana

Foreword


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It was a pleasure of going through the pages of this highly informative well-presented book entitled *Essentials of Orthopedics* written by Dr RM Shenoy whom I had the pleasure of knowing him since his childhood and later as a student and now a revered colleague.

He has an inborn dominant gene of a writer and a leader. His mother an award winning writer of short stories and poems and father a grass root social worker of repute.

I am sure this book will be a beacon of light and guide for undergraduates to understand orthopedics and the postgraduates to have their basics well forwarded and to the practicing surgeons as a ready reckoner.

I am sure this book will find a place in the shelf of every Orthopedic Surgeon.

I wish Dr Ravi all the best in his endeavors and may the good God bless him in all his endeavors.

M Shantharam Shetty MS Ortho FRCS FACS
Pro-Chancellor, Nitte University
Past President, Indian Orthopedic Association
AO Trustee 2003–2009
AO Trauma India Chairman 2009–2011
Mangalore, Karnataka, India

Good Wishes

Professor RM Shenoy is a teacher par excellence and committed to his profession. His dedication to academic activities is worth appreciating. He is an innovative Orthopedic Surgeon who is highly disciplined and meticulous in his work. He likes to improve the current status of medicine, always aiming at perfection. He expects the same from his pupils too. His original research of developing a single incision for exposure of forearm fracture speaks of his capabilities. This was published in the Journal of Bone and Joint Surgery, British 1995;77B(4):568-70. I find it to be an extremely good and a cosmetic exposure. The book *Essentials of Orthopedics* written by him is going to be a boon for both undergraduates and postgraduates as well as practicing Orthopedic Consultants. As a teacher, I am thrilled to see one of my pupils Prof RM Shenoy, reaching great heights and carving out a niche for himself among the Orthopedic Surgeons. I wish him good luck and expect many more innovative things from him in future.

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Preface to the Second Edition

The first edition of the book was written with an intent to impart basic orthopedic knowledge among medical fraternity in general and medical students in particular. The aim was to provide undergraduates with important concepts in orthopedics which would also serve as basic knowledge for postgraduates. I was indeed apprehensive about this venture, conception and execution of which took a considerable amount of time of every single day. However, it gives me great pleasure to see my efforts being well accepted and appreciated. I owe it to the medical fraternity, students and orthopedic colleagues for their support in taking this book forward to the second edition within few years of publication of the first.

With newer editions of any book, comes an unspoken requirement and responsibility. It is that of updating deficiencies that existed in the previous edition and also incorporating current information and practices. With that in mind, a few new chapters have been included on Poliomyelitis and Cerebral Palsy. Several short topics have also been included. An effort has also been made to simplify and re-write certain topics so as to precisely and effectively convey information. This includes the addition of flow charts wherever possible. I sincerely hope that this edition fulfills expectations of every reader and provides an enjoyable reading experience.

RM Shenoy

Preface to the First Edition

Medicine today is a highly specialized science. Innovations are too many and what was held good yesterday becomes obsolete today. Newer and newer concepts continue to evolve and to keep abreast with these one should have a sound basic knowledge of the subject. The subject orthopedics has very few books which impart this basic knowledge to a medical student. The book *Essentials of Orthopedics* is written keeping this fact in mind. It aims at imparting basic orthopedic knowledge to a student of medicine who has acquired basic knowledge of pre- and para-clinical subjects. The original concepts have been highlighted and the persons who put forth these concepts have been duly recognized. Every attempt has been made to narrate the concepts in a simplified manner keeping the originality. Wherever possible illustrations have been placed to help the reader to understand the subject. Radiographs have been used wherever required so that the learning process becomes easy because of the visual effect they impart. The essential topics have been covered methodically and adequately. On the whole this book is a complete book which imparts essential basic orthopedic knowledge to a medical student. Questions have been provided at the end of each chapter for revision as well as preparation for the examination. Suggestions are most welcome.

RM Shenoy

Acknowledgments to the Second Edition

Many people have come forth with suggestions and also helped me during the revision process of this title. I have tried my best to heed to every genuine criticism in the interest of the book. Foremost among my critics and if I may say, the toughest of them all, is my own dear daughter Dr Archana Shenoy, MBBS. She took it upon her to simplify the medical jargon that I sometimes would write and dedicated a lot of her time towards thorough revision of this title. Her involvement helped me fulfill many deficiencies observed in the previous edition.

I acknowledge my colleagues in Orthopedics for using this title as a teaching tool and forwarding several useful suggestions along the way. Here, I would like to specially thank Dr Sharath Rao, D Orth, MS Orth, Professor and Head, Department of Orthopedics, Kasturba Medical College, Manipal, who pointed out the absence of information on Poliomyelitis and Cerebral Palsy in the previous edition. I owe it to his keen observation, that those topics are now part of the current edition.

I recognize the help rendered to me by my residents at Yenepoya Medical College, notably, Dr Kashif Akhtar Ahmed, MBBS, (MS); Dr Tushar Jyothi, MBBS, (MS); Dr Musheer Hussain MBBS, (MS) and Dr Mohmad Irfan Nagnur, MBBS, (MS) especially during the period of final proofreading and correction.

I would also like to thank the innumerable students whom I have taught along the way, for popularizing this title and also providing some valuable suggestions.

I would also like to acknowledge the team of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, under the able leadership of its Group Chairman, Shri Jitendar P Vij for all the help and support provided during the course of revision of this title. I recognize the efforts put by Mr Venugopal, branch manager at the Bengaluru office for active support ensuring timely communication and publication of this edition.

Last but definitely not least; I would like to recognize the cooperation and sacrifices made by my dear wife Renu. I appreciate her for being with me throughout while enduring several sleepless nights and compromised family time that ensued during the course of preparation of this edition.

Acknowledgments to the First Edition

The author being honored by the revered Chancellor of Manipal University Dr Ramdas M Pai for completing 25 years of meritorious service.

At the outset, I would like to acknowledge my alma mater Kasturba Medical College which nurtured me for 39 long years first as an undergraduate student, then as a postgraduate and finally now as its faculty. I am thankful to the illustrious Chancellor of Manipal University, Dr Ramdas M Pai who guided me and gave me an opportunity to buildup my career as a young Orthopedic Surgeon. The guidance and encouragement which I had received then has put me on a sound footing today.

I am indebted to my teachers Professor M Shantharam Shetty, the Vice-Chancellor of Nitte University and President of Indian Orthopedic Association and Professor PK Usman, the two living legends who shaped my career as a teacher and an Orthopedic Surgeon.

I am grateful to my innumerable colleagues, interaction with whom considerably improved my knowledge and to the innumerable undergraduate and postgraduate students whom I have taught during the past 27 years and in the process learnt a lot.

I am extremely grateful to my present colleagues Dr Deepak Pinto, Dr Vivek Mahajan, Dr Saurabh Bansal and Dr Harshvardhan who helped me in many ways during the preparation of this book.

I acknowledge the inspiration given by my mother Smt Padma Shenoy, a State Award winning novelist, whose literary talents perhaps I have inherited, my wife Renu for kindling the spark within

me to write a book for the benefit of the students thereby enabling me to transmit this scientific, pure and unsullied knowledge to the future generation and my daughter Archana for helping me in the proofreading and correction, as well as giving all possible help and support during completion of this task.

I also acknowledge my younger brother Dr Surendra Shenoy and sister-in-law Dr Shalini Shenoy for the help they have rendered in the preparation of this book.

And last but not least; I acknowledge the efforts of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, and its enterprising Chairman and Managing Director Shri Jitendar P Vij, the dedicated team of staff at Delhi office, and Mr Venugopal, branch manager at the Bengaluru office, who have worked with me promptly and efficiently in bringing out this book in a grand manner.

Contents

General Orthopedics

INTRODUCTION

The term "Orthopedics" is from the Greek words 'Ortho' meaning straight and 'Pedia' meaning Child. It was coined by Nicholas Andry (1658–1742) who in 1741 published *L'Orthopédie, ou l'art de prévenir et de corriger dans les enfants, les difformités du Corps*' in Paris. This work of Nicholas Andry was translated into English as Orthopedia meaning the art of correcting and preventing the deformities in children. Thus wrote Nicholas Andry 'I have found it of two Greek words, e.g. straight, free from deformity, and a child. Out of these two words I have compounded that of Orthopedia, to express in one term, the design I propose, which is to teach the different methods of preventing and correcting the deformities of children.'

But the current orthopedic practice does not limit itself to straightening a child. Today an orthopedic surgeon handles almost all the pathologies involving the musculoskeletal system, namely:

A. Congenital anomalies, e.g. congenital talipes equinovarus (CTEV), developmental dysplasia hip (DDH), etc.
B. Neoplasms, e.g. osteosarcoma, giant cell tumor, etc.
C. Traumatic conditions, e.g. fractures, dislocations and fracture dislocations.
D. Degenerative diseases, e.g. osteoarthritis, spondylosis, senile osteoporosis.
E. Infections, e.g. osteomyelitis, pyogenic arthritis, bone and joint tuberculosis, etc.
F. Metabolic and endocrinal abnormalities, e.g. rickets, hyperparathyroidism, etc.

Further subspecialties have developed such as ankle and foot surgery, surgery of the upper limb and hand, spinal surgery, arthroplasty, trauma surgery, arthroscopy, etc. **Thus, currently Orthopedics has evolved into a highly specialized subject offering a variety of treatment options from simple plaster of Paris casts to skillful and complicated surgeries.**

AXIS AND DEVIATIONS

Any deviation from the anatomical axis (Fig. 1) towards or away or rotation along the axis results in deformities. The following terminologies are used to describe these deviations in the limbs.

a. ***Varus*** means deviation towards the midline and ***Valgus*** means deviation away from the midline.
b. ***Torsion*** means rotation in its axis. If rotation occurs in an inward direction, it is known as ***Intorsion***. If the rotation occurs in an outward direction it is known as ***Extorsion***.

Thus in the limbs the deformities are described as follows:

- In the hand—manus varus and manus valgus
- In the elbow—cubitus varus and cubitus valgus
- In the hip—coxa valga and coxa vara
- In the knee—genu valgum and genu varum
- In the great toe—hallux valgus and hallux varus
- In the tibia—tibial intorsion and tibial extorsion

Anteversion (Only in the Hip Region)

Angle of inclination of the long axis of the femoral neck with reference to the transcondylar plane of the distal

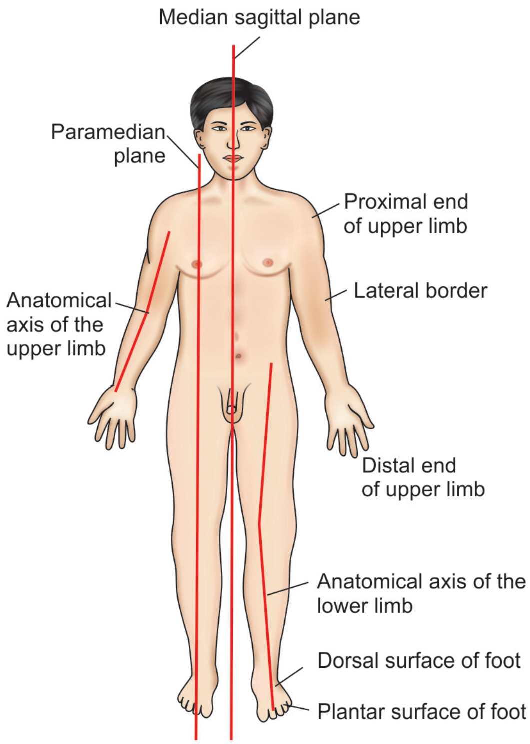

Figure 1

The anatomical position of the human body and different planes and axes.

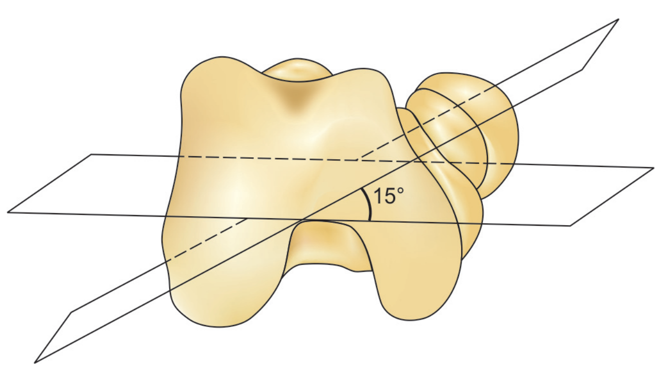

Figure 2

Normal anteversion of 15° in an adult.

femur is known as version. If this inclination is anterior it is known as ***Anteversion*** (Fig. 2) and if the inclination is posterior it is known as ***Retroversion***.

Anteversion is around 30° at birth. Progressively decreases during growth and remains at 5–15° in adults. It is always more in women than in men.

CURVATURE AND DEVIATIONS

Deviations in the Spine

The spine has normal curvatures. It is curved in a forward direction (Lordosis) in the cervical and lumbar region and in a backward direction (Kyphosis) in the thoracic and sacral region. These are the normal lordotic and kyphotic curves present in the spine. When these normal curvatures get diminished or exaggerated or bending occurs in a lateral direction, deformity develops in the spine.

Kyphosis—excessive backward (posterior) curvature of the spine.

Lordosis—excessive forward (anterior) curvature of the spine.

Scoliosis—sideward (lateral) bending of the spine.

In some of the disorders, the normal curvature may get obliterated and spine may adopt a straight posture.

Deviations in the Foot

Normal foot has an axial relation as well as a curvature. It is placed at right angles to the ankle and has an arch on the plantar aspect. Thus, the deformities may appear as a single deviation from normal or as a combination of deviations. Accordingly they are expressed using following terminologies.

Pes planus—the normal arch of the foot is lost.

Pes plano-valgus—along with planus, the foot has deviated in an outward direction (laterally).

Pes cavus—the arch is exaggerated.

Pes cavovarus—along with exaggerated arch and the foot has deviated in an inward direction (medially).

Equinus—the foot is fixed in plantar flexion.

Calcaneus—the foot is fixed in dorsiflexion.

Equinovarus, equinocavovarus and calcaneovalgus deformities (as a result of combination of deviations) are also seen. The nomenclature itself is self-explanatory.

ANKYLOSIS

Ankylosis is defined as abnormal immobility and consolidation of a joint secondary to destruction (Figs 3 and 4).


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Figure 3

An unsound bony ankylosis of the elbow as a sequel of pyogenic arthritis.

Though the ankylosis is bony and free of pain it is considered unsound because the fusion has not occurred in a functional position of the elbow which is 70° of flexion.

Figure 4

Arthrodesis of knee joint.

Causes

i. Trauma
ii. Infection
iii. Prolonged immobilization
iv. Disuse

Types

a. *Fibrous ankylosis:* In this type of ankylosis, fibrous tissue bridges the destroyed portion of the joint. Movement is possible but is painfully restricted.
b. *Bony ankylosis:* Here bone bridges the destroyed articular surfaces. The joint is completely fused and no movement is possible. The ankylosis is painless.
c. *Sound ankylosis:* Bony ankylosis in functional position is known as sound ankylosis.
d. *Unsound ankylosis:* Either fibrous ankylosis or bony ankylosis in deformed position is known as unsound ankylosis (Fig. 3).
e. *True or intra-articular ankylosis:* The stiffness occurs because of changes which have taken place inside the joint resulting in damage to the articular cartilage.
f. *False or extra-articular ankylosis:* The stiffness of the joint is due to the fibrosis and contracture of the soft tissues that surround the joint and not because of damage to the articular cartilage.

ARTHRODESIS

Surgical fusion of a joint is known as arthrodesis (Fig. 4).

Indication

A destroyed, degenerated, deformed and painful joint without function, where relief of pain and achieving stability is a prime concern, is the indication for fusion.

Types of Arthrodesis

i. Intra-articular
ii. Extra-articular
iii. Combined intra- and extra-articular

Position of Arthrodesis

The joint is fused in a position of maximum function. This position is known as functional position of a joint. It varies from joint to joint, e.g.

- Hip: 5–15° flexion, 10° abduction, 0–5° external rotation.
- Knee: 180° extension.
- Ankle: 90° extension (0° between dorsiflexion and plantar flexion) to 5° plantar flexion in women.
- Elbow (unilateral): 70° flexion.

- Elbow (bilateral): 70° flexion of dominant elbow and 110° flexion of the other (non-dominant)
- Wrist: 10–15° dorsiflexion and 5° ulnar deviation.

Examples of Arthrodesis

i. *Blair's fusion* of the ankle for old fracture neck of the talus with avascular necrosis (AVN).
ii. Wrist arthrodesis for AVN and nonunion of the scaphoid fracture with painful degenerative arthritis.

ARTHROPLASTY

Arthroplasty is a procedure by which a destroyed, degenerated, deformed, painful joint is rendered painless along with restoration of its lost movement.

Types

a. *Excision arthroplasty:* The diseased portion is removed and a false joint is allowed to form, e.g. Girdle stone procedure for tuberculosis (TB) hip.
b. *Interposition arthroplasty:* A muscle or fascia is interposed between the two articular surfaces in order to promote painless movements e.g. fascia lata interposition in elbow arthroplasty.
c. *Hemiarthroplasty:* One-half of the joint is replaced. Other half is left alone e.g. Austin Moore's prosthetic replacement of head and neck of the femur in fracture neck of the femur. The acetabulum is untouched.
d. *Total joint arthroplasty:* Replacement of both the articular surfaces with an artificial prosthesis, e.g. a total hip replacement, a total knee replacement.

ORTHOPEDIC SPLINTS

Splint is a device used to externally support and to give rest to the limb or an appendage. They are made of various materials. These days, polymers of plastic are being used in a big way to prepare various splints.

Static Splints

These splints only support and give rest to the part, e.g. cock-up splint, posterior gutter splint, etc.

Dynamic Splints

These splints not only give support and rest to the part but also assist and allow movement of the recovering as well as uninjured structures e.g. knuckle bender splint, dynamic wrist drop splint, etc.

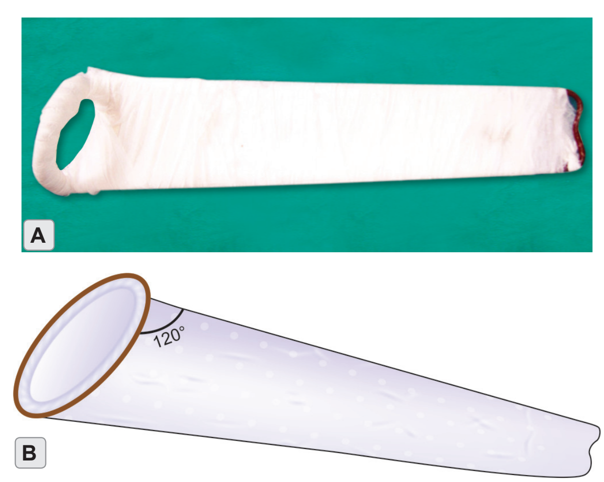
Specific Types of Splints

Thomas' Splint (Figs 5A and B)

It is a splint described by Sir Hugh Owen Thomas. It was initially used to immobilize the knee joint in tuberculosis of the knee. Hence was known as Thomas' bed knee splint.

Design of the Thomas' Splint

- *Lower limb:* It has a ring and two side bars. These days the side bars are welded to the ring, right in the middle of the circumference, bisecting it into two equal halves. This makes it suitable for application either to the left or to the right lower limb. The ring is placed at an angle of 120° to the inner bar. The side bars are not parallel. They converge towards the foot end where they are bent in the shape of a 'V' for the attachment of a traction cord.
- *Upper limb:* The splint has a smaller ring and the ring is placed at right angles to the bars (not at 120°).



Figures 5A and B
Thomas' splint. Note the convergence of the side bars, placement of the ring which is at 120° to the inner bar and for universal use, the bars are welded to the ring in the middle bisecting it into two equal halves. **Note:** *Originally the side bars were welded to the ring at the junction of anterior 2/3 and posterior 1/3 to accommodate the girth of the thigh. So, there were separate splints for right and left lower limb.*

Measurement of the ring: The oblique circumference at the groin is measured below the gluteal fold, crossing the ischial tuberosity to the anterosuperior iliac spine. Then 4 inches are added to this circumference.

Measurement of the length: 4–6 inches more than the length of the limb.

Application: A proper sized splint has to be used for giving desirable support. Adequate padding of the splint and taking care to avoid pressure points is a must.

Uses of Thomas' Splint

i. In the first-aid treatment of the fracture of the lower limbs.
ii. In children for the definitive treatment of fracture femur.
iii. In the temporary immobilization of lower limb for shorter periods, e.g. postoperative immobilization.

Bohler-Braun Splint (Fig. 6)

This splint bears the name of two people Bohler and Braun. It has two components. A lower component on which the lower limb rests, i.e. Braun splint and an upper component to which a set of 3 pulleys are attached at strategic places for different applications i.e. Bohler's modification.

Uses of Bohler-Braun splint

i. In the definitive treatment of lower limb fractures by skeletal traction.
ii. As a temporary support and for elevation of the limb.

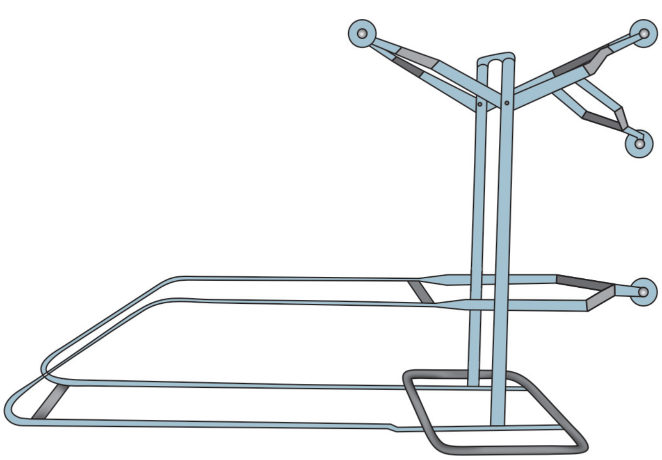


Figure 6

Bohler-Braun splint

Showing the lower portion (frame) on which the limb rests and to which is attached at right angles a U stirrup with a system of Pulley's.

TRACTIONS

The meaning of the word traction is to pull.

Types of Traction

a. *Skin or surface traction:* The skin is used as a medium for application.
b. *Skeletal or bone traction*: The bone is used as a medium for application.

Methods of Traction

a. *Fixed traction:* In this, no weights are suspended. The traction unit is fastened to the splint at its end.
b. *Sliding or the balanced traction:* In this, weights are suspended to the traction unit. These weights act as a force. One end of the cot (either foot end or head end) is raised so that the body weight acts as a counter force. Between these two forces, namely the weight suspended on one side and the body weight on the other, the fracture maintains the desirable position of reduction that is achieved.

Skin Traction (Fig. 7)

It is one of the well accepted methods of giving traction. In the past adhesive tapes were used. Because of skin irritation these are now given up. Traction units made of foam and latex rubber are readily available for use.

Application: The traction units are tied to the limbs with the help of bandages and weights are suspended (sliding traction) or a fixed traction is given on a splint. Maximum weight which can be used for skin traction is 5–7 kilograms.

Uses of skin traction

i. Temporary immobilization and immobilization for a shorter period.
ii. Treatment of pediatric fractures, e.g. Gallows traction.
iii. Treatment of prolapsed intervertebral disk.

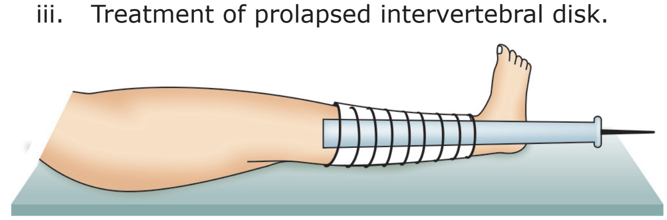


Figure 7

Skin traction applied to the lower limb. In this, only below knee traction is shown. When needed an above knee traction can also be applied.

Skeletal Traction

Devices used: (Fig. 8)

- In adults
  - Steinmann pin and Bohler's stirrup and in lower limbs.
  - Kirschner's wire and Kirschner's wire tensioniser or stretcher in upper limbs.
- In children
  - Kirschner's wire and Kirschner's wire tensioniser.
- In elderly with osteoporosis
  - Denham's pin with Bohler's stirrup (See Chapter on Instruments).

Sites of skeletal traction

i. Calcaneum.
ii. Lower end of the tibia.
iii. Upper end of the tibia (Fig. 9).
iv. Lower end of the femur.
v. Greater trochanter.
vi. Through the metacarpals.
vii. Through the olecrenon.
viii. Skull traction.

Uses of skeletal traction

i. Immobilization for a longer period.
ii. Preoperative distraction.
iii. Definitive treatment of fracture.

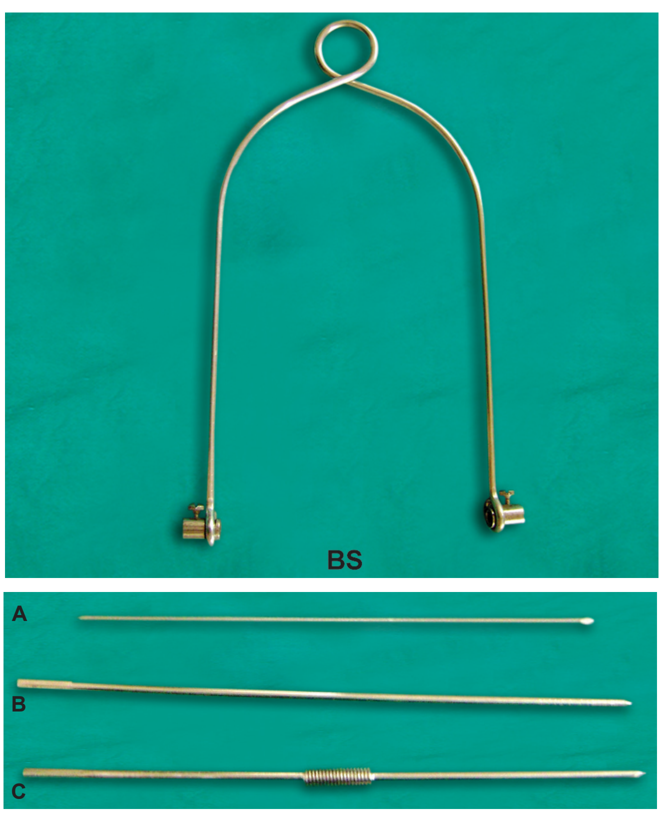


Figure 8

BS—Bohler's stirrup, A—'K' wire, B—Steinmann pin, C—Denham's pin.

Note: Pre operative distraction is necessary to keep the fracture ends apart and prevent overriding of fragments when there is delay in definitive treatment. This makes the later reduction simple in closed reduction and fixation of fractures.

Skull Traction

Skull traction is given for cervical spine injury, i.e. in cases of fractures, dislocations and fracture dislocations of the cervical spine.

Devices used: Crutchfield tongs, Barton's tongs, Gardner Well's tongs, etc. (Figs 10 and 11).

Complications of skeletal traction

i. Infection and osteomyelitis.
ii. Loosening and cut through.
iii. Nerve palsies (in some areas because of faulty insertion or positioning the limb after traction).
iv. Splintering of bone (unskilled insertion).

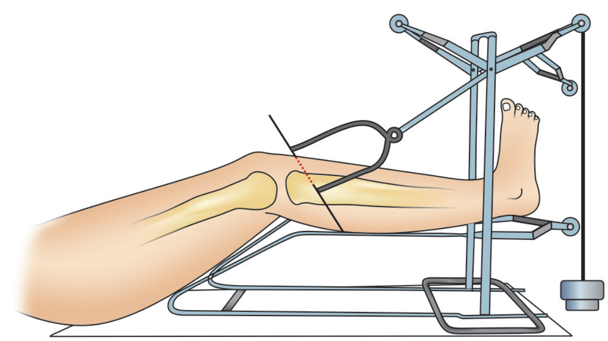


Figure 9

Skeletal traction through the upper end of the tibia using the Steinmann pin and the Bohler's stirrup.

Figure 10

Crutchfield tongs used for skull traction.

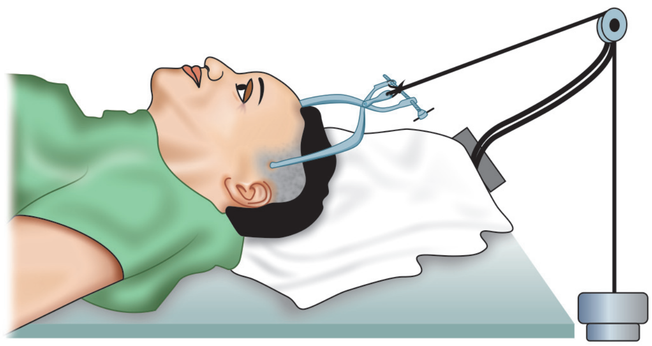


Figure 11
Application of skull traction.

OSTEOTOMY

Surgically cutting the bone under direct vision using an osteotome (an instrument which is used to cut the bone) is known as osteotomy. These days the power saw is used for the same. The procedure is indicated for correction of deformities in:

1. Malunion of fractures, e.g. malunited distal radius fracture.
2. Osteoarthritis, e.g. a high tibial osteotomy.
3. Genu valgum of rickets.
4. Osteotomies for Developmental Dysplasia of Hip (DDH).

OSTEOCLASIS

Manually breaking the bones (intentional) without opening the site is known as osteoclasis. This procedure is done under anesthesia for correction of deformities in:

1. Greenstick fractures.
2. Early phase of fracture union when the callus is soft.

OSTEOSYNTHESIS

The method of stabilizing fractures using implants is known as osteosynthesis. Implants act as an internal splint e.g. plates, screws and nails. Different designs of plates, e.g. LC DCP (limited-contact dynamic compression plate), LCP (locking compression plate), 'T' plates, 'L' plates, etc. nails, e.g. titanium elastic nails, ender nails, interlocking nails, proximal femoral nails, etc. screws, e.g. cortical screw, cancellous screw, malleolar screw, Herbert screw, etc. are available for osteosynthesis and are used as per indication.

TYPES OF ARTICULATIONS (JOINTS) IN THE HUMAN BODY

The three basic types of articulations (joints) seen in the human body are:

Fibrous Articulation (Joints)

Suturous

In this type the sutural membrane exists between the bones, e.g. in the skull. Subsequently the two bones fuse, the membrane disappears and a synostosis develops (Fig. 12).

Syndesmotic (Syndesis—Bound Together)

The bony components are held (bound) either by interosseous ligaments at the end or by interosseous membrane along its surface, e.g. inferior tibiofibular syndesmosis and radioulnar syndesmosis (Figs 13A and B).

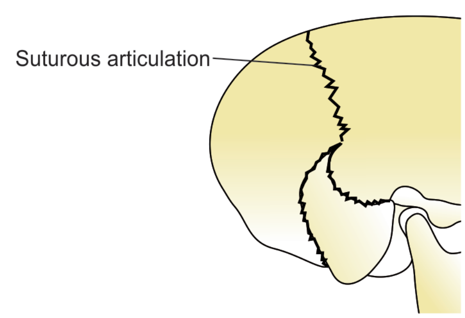
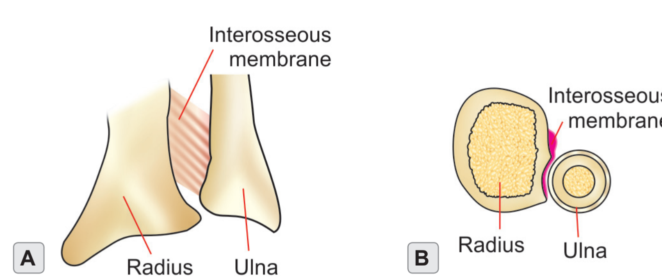


Figure 12
Suturous articulation.



Figures 13A and B
Syndesmotic articulation.

Cartilaginous Articulation (Joints)

Synchondrosis (Syn—Together, Chondros—Cartilage: Meaning Joined Together with Cartilage)

Seen in a growing bone and in pediatric age group. The intervening hyaline cartilage of this articulation disappears with growth, e.g. junction of epiphysis with diaphysis (Fig. 14).

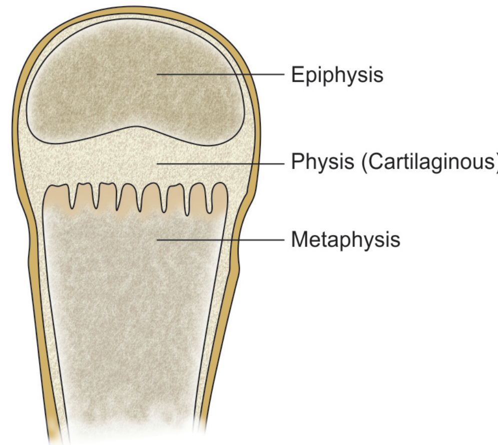
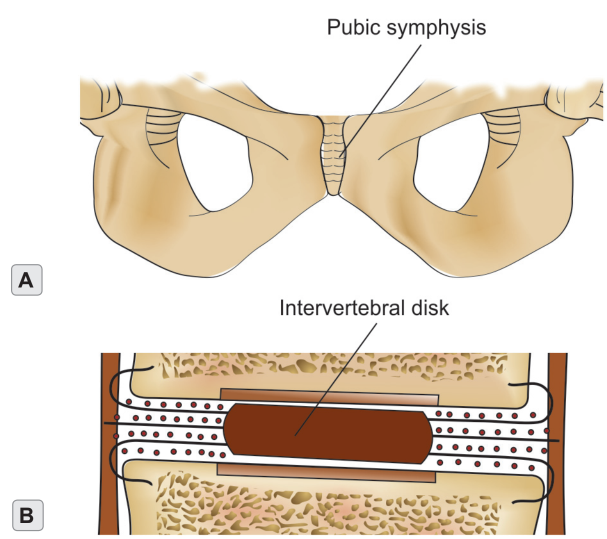


Figure 14
Synchondrosis.

Amphiarthrosis (Amphi—On Both Sides, Arthro—Articular: Meaning Articulation on Both Sides)

In this type of articulation the ends of the bone are covered with articular cartilage and a fibrocartilagenous disk exists between them. This intervening disk binds them as well as the periosteum of the bone. Such articulation is seen in the median plane, e.g. pubic symphysis, between the two vertebral bodies in the spinal column (Figs 15A and B).



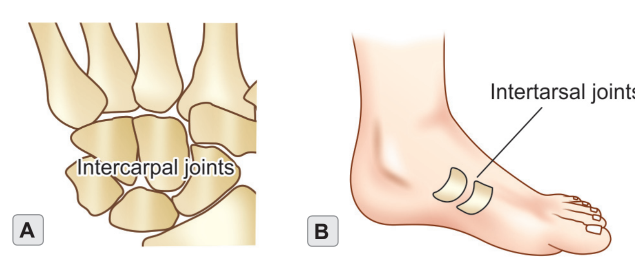
Figures 15A and B
Amphiarthrosis.

Synovial Articulation (Joints)

This type of articulation is seen mainly in the limbs. The articulation is known as diarthrosis and the joint is known as diarthrodial or synovial joint. In a synovial articulation, the ends of the bones are covered with articular cartilage and the whole structure is enclosed within a fibrous capsule which is continuous with the periosteum on either side. The inner surface of the capsule as well as the areas of bone and the intra-articular structures which are not covered by articular cartilage are lined by a synovial membrane. This synovial membrane secretes synovial fluid which lubricates the joint. A good range of movement exists in these joints. Hence, these joints are supported by ligaments which give additional stability.

The different types of synovial articulation are as follows:

i. Gliding or arthrodial, e.g. intertarsal and intercarpal articulation. A gliding movement (translation) takes place in these joints. The articular surfaces are neither concave nor convex. They are flat and conducive for gliding (Figs 16A and B).
ii. Hinge or Ginglymous (Greek—means hinge), e.g. humeroulnar articulation of the elbow joint. A unidirectional movement takes place in this articulation along a transverse axis, i.e. flexion and extension (Fig. 17).



Figures 16A and B
Gliding joints.

iii. Condyloid, e.g. wrist joint, temporomandibular joint. This is a shallow articulation between a condyle and an elliptical concave surface. A wide range of movement takes place in these joints except rotation in its axis (Fig. 18).

iv. Pivot or Trochoid (Greek—Trochos means wheel), e.g. superior radioulnar joint, atlantoaxial joint. A rotational movement takes place around a pivot in these articulations (Fig. 19).

v. Saddle or Sellaris (Latin—Sella means Saddle), e.g. carpometacarpal joint of the thumb. In this articulation a wide range of movement takes place between the two articular surfaces, which are individually concavo-convex and correspond with each other (Fig. 20).

vi. Ball and socket or enarthrosis (Greek—En means In), e.g. shoulder and hip joint. A wide range of movement, unidirectional as well multidierctional; single or in combination occurs in this joint including rotation in its axis (Fig. 21).

vii. Combined type, e.g. knee joint and ankle joint. These joints have properties of Hinge (maximum) as well as pivot and gliding (minimum), respectively (Fig. 22). In the knee, after complete extension the tibia rotates externally like a pivot joint and joint gets locked. In the ankle joint, minor degrees of gliding takes place in extremes of dorsiflexion and plantar flexion. This happens because of inherent design of the articular surface.

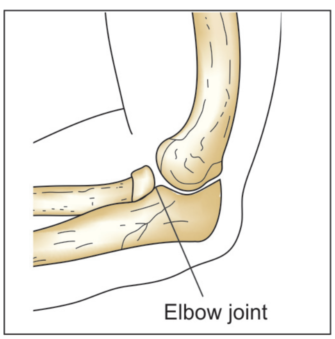


Figure 17
Hinge joint.

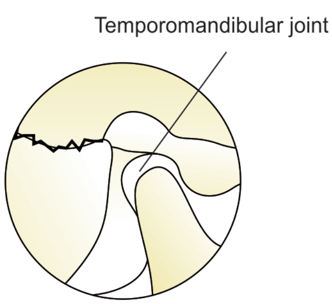


Figure 18
Condyloid joint.

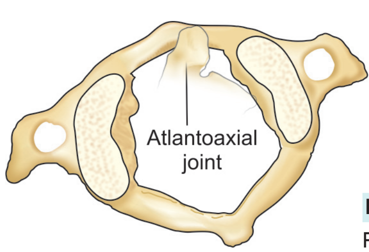


Figure 19
Pivot joint.

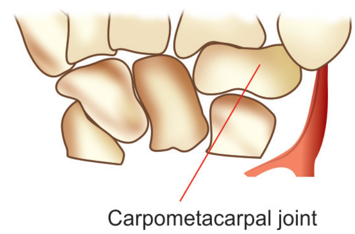


Figure 20
Saddle joint.

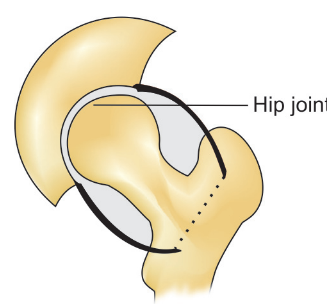


Figure 21
Ball and socket joint.

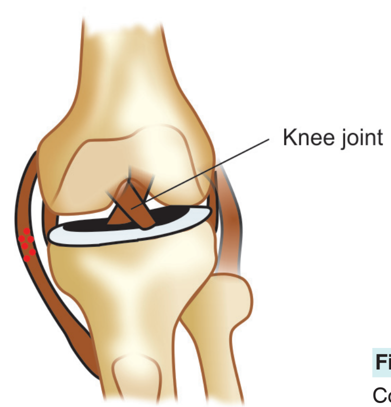


Figure 22
Combined type.

GENERAL ABBREVIATIONS

#	Fracture
ABC	Aneurysmal Bone Cyst
ACL	Anterior Cruciate Ligament
AFO	Ankle Foot Orthosis
AK	Above Knee
AMP	Austin Moore Prosthesis
ANA	Antinuclear Antibody
AS	Ankylosing Spondylitis
AVN	Avascular Necrosis
BK	Below Knee
BMP	Bone Morphogenic Protein
CMC	Carpometacarpal Joint
CRP	C-Reactive Protein
CT	Computerized Tomography
CTEV	Congenital Talipes Equinovarus
CTS	Carpal Tunnel Syndrome
DCP	Dynamic Compression Plate
DDH	Developmental Dysplasia Hip
DHS	Dynamic Hip Screw
DIP	Distal Interphalangeal Joint
DRUJ	Distal Radioulnar Joint
DTR	Deep Tendon Reflexes
EMG	Electromyography
ESR	Erythrocyte Sedimentation Rate
FWB	Full Weight-Bearing
GCT	Giant Cell Tumor.
HME	Hereditary Multiple Exostosis
HNP	Herniated Nucleus Pulposus
IL	Interlocking
IM	Intramuscular
IM	Intramedullary
IP	Interphalangeal
IT	Iliotibial
IVDP	Intervertebral Disk Prolapse
LC DCP	Limited Contact Dynamic Compression Plate
LCP	Locking Compression Plate
LISS	Less Invasive Stabilization System (Acronym)
LP	Lumbar Puncture
MCL	Medial Collateral Ligament
MCP	Metacarpophalangeal (Joint)
MED	Multiple Epiphyseal Dysplasia
MIPPO	Minimally Invasive Percutaneous Plate Osteosynthesis (Acronym)
MRI	Magnetic Resonance Imaging
MTP	Metatarsophalangeal (Joint)
NSAID	Nonsteroidal Anti-inflammatory Drug
OA	Osteoarthritis
OI	Osteogenesis Imperfecta
ORIF	Open Reduction Internal Fixation
OS	Osteosarcoma
PCL	Posterior Cruciate Ligament
PET	Positron Emission Tomography
PIP	Proximal Interphalangeal (Joint)
PSA	Prostate Specific Antigen
RA	Rheumatoid Arthritis
RF	Rheumatoid Factor
ROM	Range of Motion
SCFE	Slipped Capital Femoral Epiphysis
SLE	Systemic Lupus Erythematosus
SPECT	Single Photon Emission Computed Tomography
TENS	Transcutaneous Electrical Nerve Stimulation
TFCC	Triangular Fibrocartilage Complex
THR	Total Hip Replacement
TKR	Total Knee Replacement
TMJ	Temporomandibular Joint
UBC	Unicameral Bone Cyst
VIC	Volkmann's Ischemic Contracture

COMMON SYNDROMES IN ORTHOPEDICS

Albright's Syndrome

Characterized by, polyostotic fibrous dysplasia, café-au-lait spots and precocious sexual puberty.

Maffucci's Syndrome

Characterized by multiple Enchondromata, soft tissue hemangiomas in the skin and the viscera with dyschondroplasia.

von Recklinghausen's Disease

Characterized by multiple cutaneous neurofibromata, café-au-lait spots and musculoskeletal abnormalities, e.g. scoliosis. It is autosomal dominant.

Nail Patella Syndrome

Characterized by hypoplastic nails, unusually small patellae, subluxated head of the radius, pelvic horns and congenital nephropathy. It is autosomal dominant.

Morquio-Brailsford Disease

Characterized by dwarfism, moderate kyphosis, short neck, protuberant sternum, increased joint laxity and genu valgum. It is autosomal recessive.

Frolich Syndrome

Associated with slipped capital femoral epiphysis. Characterized by delayed skeletal maturity with poor secondary sexual characters and adiposity (obesity).

Fanconi's Syndrome

Characterized by renal rickets with glycosuria, phosphaturia and aminoaciduria. It is autosomal dominant.

1 Introduction to Fractures and Dislocations

- ☞ *Definition of a fracture, types, healing and treatment of fractures*
- ☞ *Complications and their treatment*
- ☞ *Recent concepts in fracture healing*
- ☞ *Definition of dislocation, types, management in general and complications*

Fractures and dislocations are among the most common injuries seen in day-to-day practice. It is important to remember the following basic facts to understand these injuries.

A. A force of considerable magnitude is necessary to cause these injuries (Unless the bone is already weak or the structure of a joint is already disturbed due to disease).
B. The resultant failure pattern (deformation) is directly proportional to the nature, magnitude and direction of the force.
C. It is possible to classify these failure patterns with certain limitations.
D. The treatment protocol is based on the nature of the failure pattern and follows a definite path which is consistent, with minor variations.
E. Healing is inversely proportional to the severity of the injury.
F. Complications that develop with these injuries are related to the severity of the deforming force, the resultant failure pattern, the site and multiplicity of the injury.
G. Though the terms 'Fracture and Dislocation' refer to the bone and joint pathology, one should always remember that in a skeletal injury, there is considerable damage to the soft tissue envelope that surrounds the bones and the joints i.e. periosteum, muscles, ligaments, tendons and so on. This soft tissue injury has an inverse relation to the normal healing process.

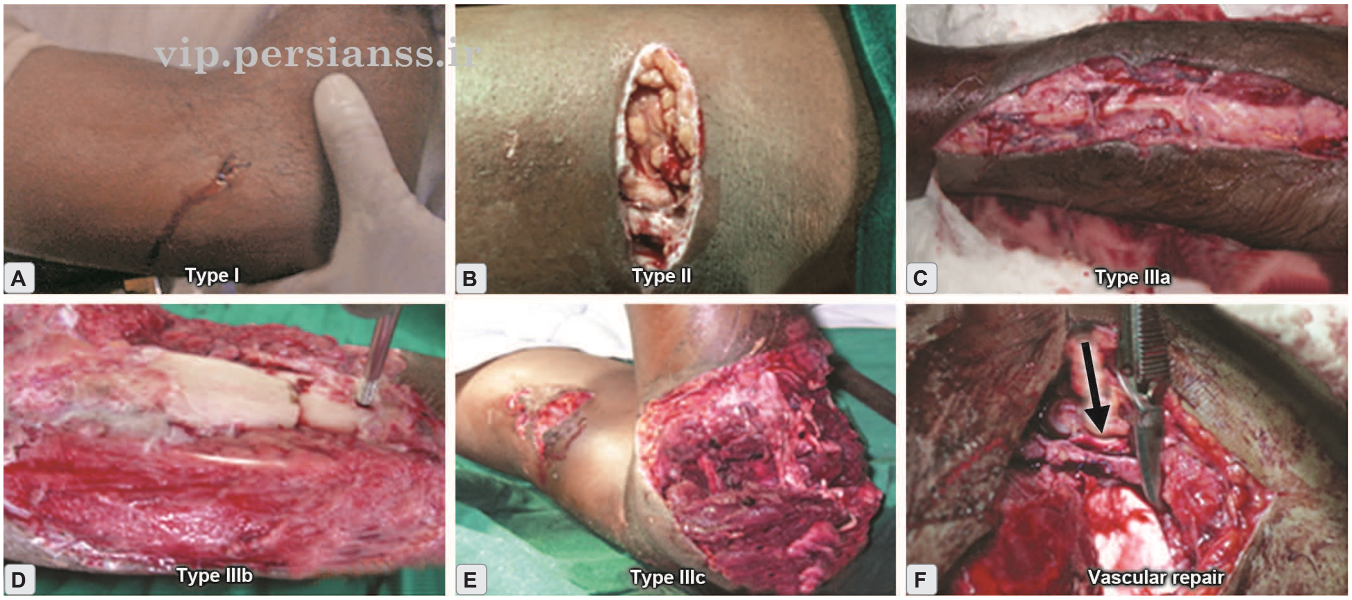
FRACTURES

Definition

Fracture can be defined as a break in continuity of a bone or the loss of normal anatomical continuity of a bone.

Types of Fracture

I. Depending on nature
   a. Closed or simple: The fracture site does not communicate with the exterior.
   b. Open or compound: The fracture site communicates with the exterior (Figs 1.1A to F).
II. Depending on the displacements
   a. Displaced fracture.
   b. Undisplaced fracture
      – Incomplete fracture.
      – Complete fracture.
III. Depending on the nature of the fracture line (Figs 1.2 to 1.8)
   a. Transverse fracture.
   b. Oblique fracture
      – Long oblique.
      – Short oblique.



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Figures 1.1A to F

Classification of open fractures—based on the classification by Gustilo and Anderson.

Criteria for classification: Extent of injury to skin, soft tissue, bone, vessels and the degree of contamination.

Type I: Wound smaller than 1 cm in diameter, no skin crushing with no or little contamination. Fracture pattern is not complex

Type II: A lacerated wound larger than 1 cm without significant soft tissue crushing or contusion, no degloving with moderate contamination. Fracture pattern may or may not be complex.

Type III: An open injury with extensive soft tissue crushing and contamination, fracture pattern is single or complex. Injury is further subdivided into three types:

a. Adequate soft tissue coverage of the fracture can be acquired at closure.

b. Periosteal stripping is seen. Inadequate soft tissue coverage at closure. Hence, soft tissue reconstruction is necessary.

c. Open fracture that is associated with vascular injury (or nerve injury or both) and that needs repair.

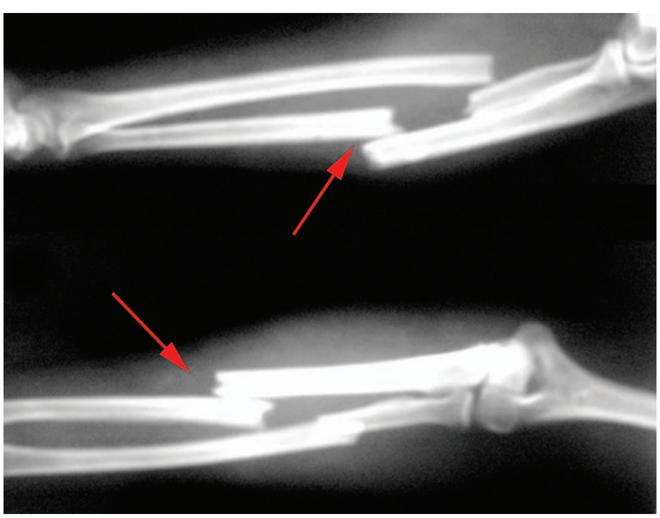


Figure 1.2

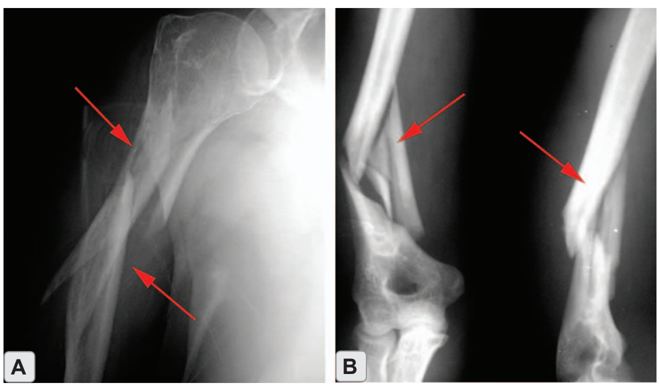
Fracture of radius and ulna. Note the transverse nature of the fracture line.

Figure 1.3

A short oblique fracture at the neck of the 5th metacarpal bone of the left hand.

Figure 1.4

A short spiral fracture of the tibia and the fibula, in the lower third.



Figures 1.5A and B

Fracture of upper and lower humerus respectively and comminution at the fracture site with many fragments of bone which are displaced.

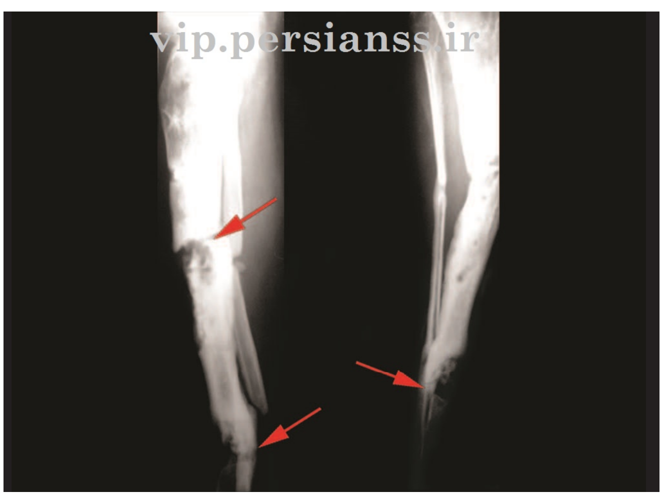

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Figure 1.6

An open segmental fracture of tibia and fibula with secondary infection and changes of chronic osteomyelitis. Bone resorption is seen at the fracture site. Both the fracture sites are showing features of nonunion.

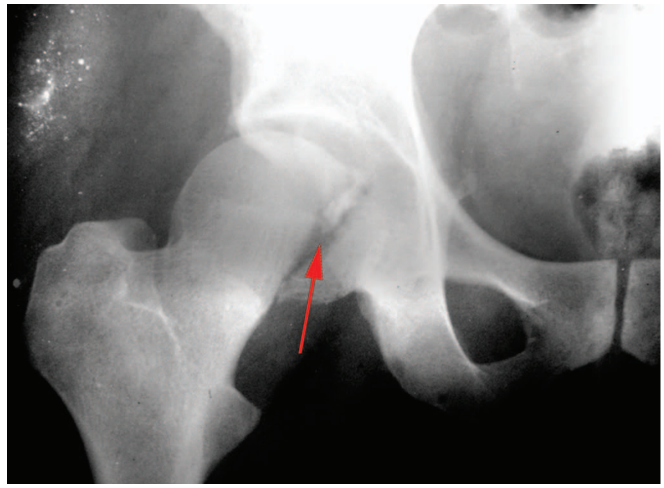

Figure 1.7

Fracture of the head of the femur. This is a very rare injury and generally associated with a dislocation of the hip. In this case, there was posterior dislocation which was reduced. The reduction achieved is obviously not congruous.

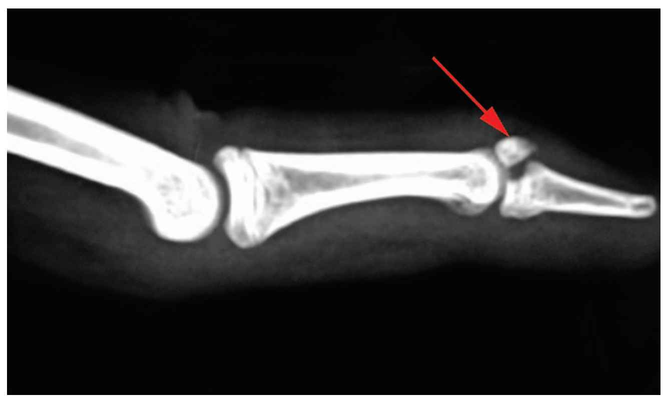

Figure 1.8

An avulsion fracture of the base of distal phalanx. The resultant deformity is a 'Mallet finger'. The patient is a goal keeper and the injury happened during a soccer match when he blocked a penalty kick. Avulsion force is exerted by the lateral slips of the extensor expansion.

c. Spiral fracture
   - Long spiral.
   - Short spiral.
d. Comminuted fracture: In this type, there are more than two fragments at one fracture site.
e. Segmental fracture: In a single bone, fracture occurs at two different levels.
f. Intra-articular fracture: A fracture that involves (extends into) the articular surface of a joint.
g. Avulsion fracture: This is a fracture occurring due to a pull by a muscle, tendon or a

ligament at its insertion to the bone, e.g. mallet finger, fracture of the olecranon process of the ulna.

Note: *Multiple fractures is the terminology used when many bones are fractured in an individual. This should not be confused with segmental or comminuted fracture (see above).*

Deforming Forces

The deforming forces causing fractures can be classified based on impact as:

a. Direct
b. Indirect

Direct impact causes severe injuries, e.g. open fractures, comminuted fractures, etc.

Indirect impact causes less severe injuries. Forces that cause indirect impact are classified as follows:

i. A bending force—produces a transverse fracture.
ii. A torsional force—produces a spiral fracture.
iii. A combination of both bending and torsional force—produces a comminuted fracture with a butterfly fragment/fragments.

Specific Types of Fracture

Greenstick Fracture (Fig. 1.9)

The fracture got its name because the bone breaks like a greenstick branch of a tree. Only a part (one side) of the bone breaks and rest of the bone bends. It is an incomplete break occurring in the bone and seen in children whose bones are more elastic, soft and pliable. There is no abnormal mobility.

Compression Fracture (Fig. 1.10)

It is seen in the vertebral column wherein the height of the vertebral body decreases following fracture.

Figure 1.9
Greenstick fracture of both, the radius and the ulna. Note the gross deformity.

Pathological Fracture (Fig. 1.11)

It is a fracture occurring in a bone with pre-existing pathology. The pathology softens the bone considerably and this soft bone yields to a very trivial trauma and fractures, e.g. malignancy, osteomyelitis, etc.

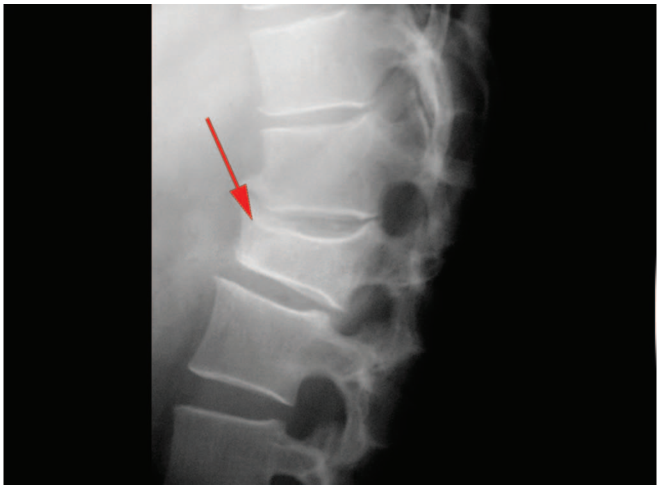

Figure 1.10
A compression fracture of L2 vertebra. Note the decrease in anterior height of the vertebral body.


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Figure 1.11
A Giant cell tumor arising from the upper end of tibia with multiple pathological fractures due to thinning of the cortex. Pathological fracture is a late feature of a Giant cell tumor.

Stress Fracture (Fig. 1.12)

It is a type of pathological fracture due to unaccustomed stress getting concentrated on one part of the bone, e.g. March fracture seen in soldiers after a long route march.

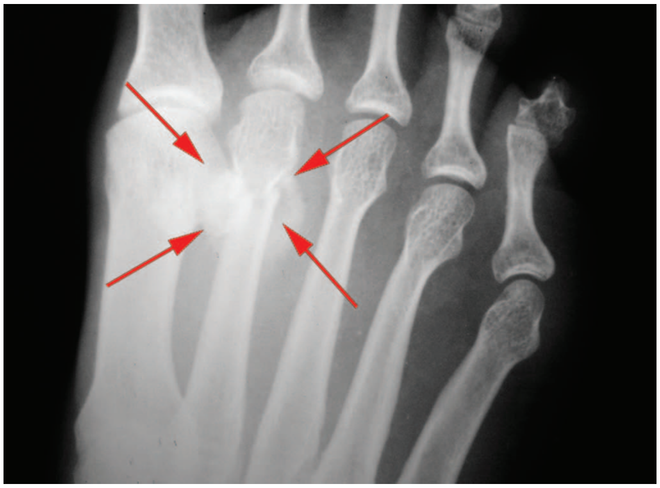


Figure 1.12

'March fracture' at the neck of 2nd metatarsal. Note the healing response by formation of abundant callus.

Signs and Symptoms of a Fracture

1. Pain and swelling.
2. Deformity.
3. Loss of continuity.
4. Irregularity.
5. Crepitus.
6. Bony tenderness.
7. Loss of function.
8. Abnormal mobility.

(Abnormal mobility is a sign to be observed and not a sign to be elicited in a fresh fracture. It is very painful because the movement is taking place at the fracture site. It is the sure sign of a fracture and when present further examination to ascertain the presence of a fracture is unnecessary.)

Healing of a Fracture

Healing takes place in stages and over a period of time which is said to be a minimum of 4 weeks approximately. Four distinct stages are recognized (Fig. 1.13).

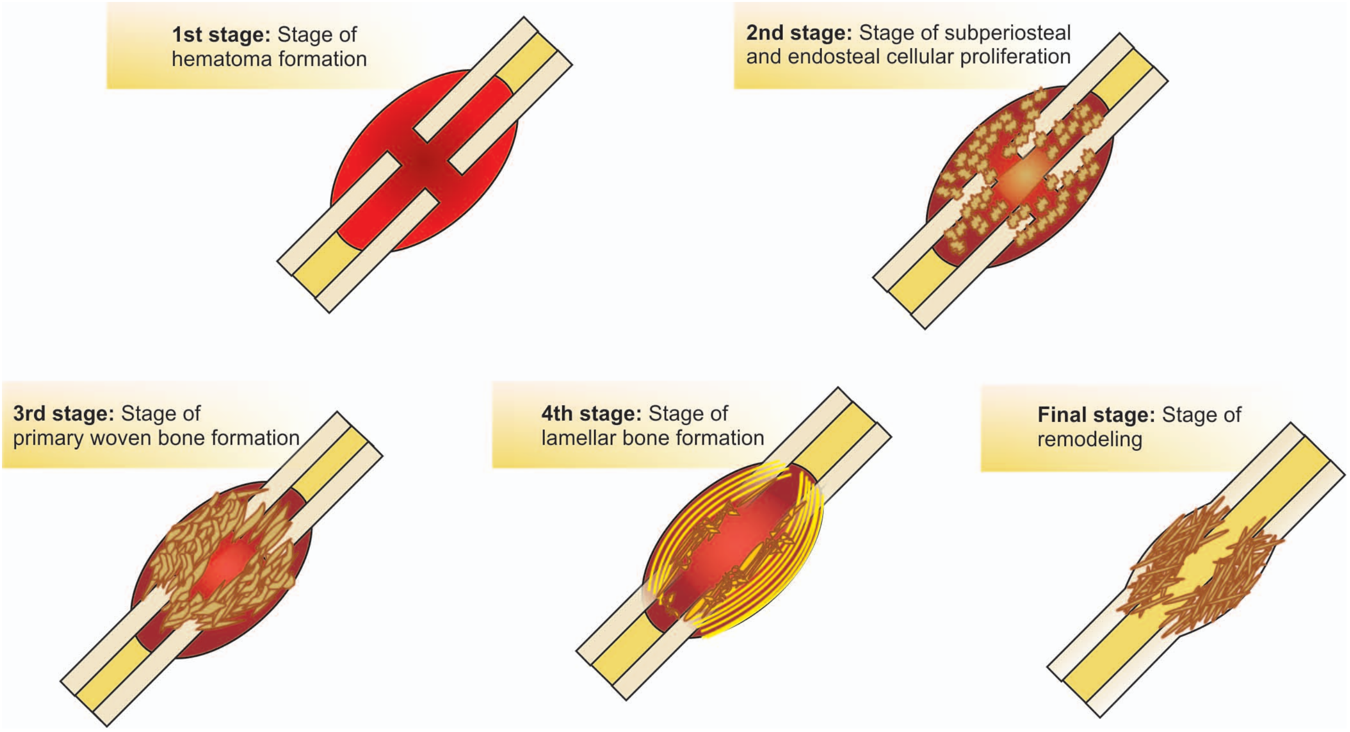


Figure 1.13

Stages of healing of fracture.

1st Stage: Stage of Hematoma Formation

This is an important stage of fracture healing. As the bone fractures, the blood vessels are torn and hence bleeding occurs almost immediately. Hematoma acts as a vehicle delivering the required material for union and clearing unwanted material by a process of chemotaxis of cells. If this stage of hematoma is deficient as seen in cases of open fractures, healing is interfered with and fracture fails to unite.

(Hence open fractures are to be converted into closed fractures as early as possible to promote healing. Many a times this is not possible and *the resultant nonunion is accepted and treated accordingly at a later date.)*

2nd Stage: Stage of Cellular Proliferation

Within 8 hours of fracture there is inflammation subsequently leading to subperiosteal and endosteal cellular proliferation. These cells surround the broken ends of the bone. At the same time, the clotted hematoma progressively gets absorbed and new capillaries start infiltrating these cellular masses.

3rd Stage: Stage of Primary Woven Bone Formation (Soft Callus)

The proliferating cells which are mainly osteogenic and chondrogenic start to get incorporated into the fibrogenic matrix under the influence of bone morphogenic proteins (BMP), the transforming growth factor beta (TGF-β) and fibroblast growth factor (FGF), thus forming primary woven bone. This bone is soft as it is not fully mineralized. This occurs during the 2nd and 3rd week.

4th Stage: Stage of Lamellar Bone Formation (Hard Callus)

Mineralization occurs and the primary woven bone is transformed into lamellar bone. This occurs between 3 and 6 weeks.

Lamellar bone is hard and is seen as a bridge or a cuff across the fracture site. It indicates an early stage of fracture union.

Stage of Remodeling

This stage is better not considered as one of the stages of fracture healing because remodeling takes place only after the fracture unites (heals) and takes months and years. Here, the body attempts to give the normal shape and strength to the fractured bone or in other words to restore its preinjured status. Remodeling is rapid in children and in growing bones, slow in adult bones and almost nil in osteoporotic bones.

Note: *Healing of a fracture in a cancellous bone does not follow these stages. Cancellous bone heals by direct formation of osteoblastic new bone.*

Factors Influencing Healing (Predisposing Factors for Delayed Union and Nonunion)

a. Factors not within the control of treating doctor
   - Nature of the trauma
     – High velocity trauma.
     – Low velocity trauma.
   - Nature of the fracture.
   - Vascularity of the bone.
   - Age of the patient.

b. Factors within some control of the treating doctor (failure to achieve these results in delayed and nonunion).
   - Proper reduction.
   - Adequate fixation.
   - Adequate immobilization.
   - Prevention of distraction.
   - Prevention of infection.
   - Maintaining adequate nutrition.
   - Adequate management of other comorbid conditions, e.g. diabetes, hypertension, osteoporosis, etc.

Management of a Fracture

The treatment begins at the site of injury. The first step is to apply a splint to the injured part. After this a thorough general and systemic examination is performed to look for other injuries such as vascular visceral and neurological. Visceral injuries when present may be life-threatening, e.g. liver laceration, splenic rupture, hemopneumothorax, etc. Early repair of these structures is of paramount importance. In polytrauma, maintaining the airway, breathing and blood pressure (by treating of shock and hemorrhage) is essential. A skilled paramedical team should be available to transport a severely injured patient to a specialized center where definitive treatment is instituted. Delay can be detrimental.

Basic Methods of Treating a Fracture

1. Immobilization in a cast.
2. Closed reduction and immobilization in a cast.
3. Open reduction and internal fixation.
4. Closed reduction and internal fixation (with the help of C-Arm imaging).
5. External fixation.
6. Tractions.

All the above methods are practised and can be selectively employed to treat a fracture depending on the indication. All these methods help to enhance the biological process of healing by maintaining the anatomical alignment and providing necessary stability at fracture site. Every method has its own merits and demerits and is to be chosen carefully. Aim of fracture treatment is to minimize the confinement to bed, achieve union at the earliest and make the patient regain his activity as early as possible.

Steps of Managing an Open Fracture

Management of open fracture is an emergency and has to be carried out precisely. Apart from imparting primary care for the open wound, it is important to maintain vital parameters and attend other associated injuries (when present).

- Patient is examined as a whole for other associated injuries, e.g. head injury, chest and visceral injury. His general condition is also ascertained.
- IV fluids and broad spectrum antibiotics are administered for maintaining the vital parameters and preventing infection. At the same time blood is drawn for relevant investigations, e.g. Hb%, packed cell volume (PCV), blood group, HbSAg, etc.
- Immediate care of the wound is given in the form of saline or clean water wash and application of sterile compression dressing to minimize oozing.
- Immediate immobilization of the injured part is done with an appropriate splint.
- If the patient is in a state of shock all resuscitative measures are employed immediately. Excessive bleeding is managed by blood transfusion. If blood is not available, plasma expanders are used as a temporary measure.
- If shifting to a better center becomes necessary, patient is transported only after he is stabilized and in a well equipped ambulance along with a medical team.
- Definitive management is carried out only in a well equipped center.

Definitive Treatment of Open Fractures

As soon as the patient is stable and fit for surgical intervention definitive treatment is instituted. Aim of treatment is to convert a contaminated wound into a clean wound and an open fracture into a closed fracture as early as possible.

- Wound debridement is done under anesthesia by clearing all contaminants, excising all dead, devitalized tissues and contaminated bone upto clean bone.
- The fracture is stabilized using external fixator or intramedullary solid nails as per indication.
- The wound is sutured in order to convert an open fracture into a closed fracture at earliest or a flap coverage is done primarily when feasible.
- When not feasible a secondary closure is done as early as possible.

Severe grades of open fracture may go for nonunion and delayed union which are managed subsequently with secondary procedures such as bone grafting, distraction compression osteogenesis, etc.

Complications

Following are the complications that can occur after a fracture.

A. *Specific and local complications*

Early
1. Associated nerve injuries.
2. Associated vascular injuries (Fig. 1.14).
3. Associated visceral injuries.

Delayed
1. Nonunion
2. Delayed union
3. Malunion
4. Infection leading to osteomyelitis and pyogenic arthritis (as a consequence of open fracture or surgical sepsis).

B. *Systemic and general complications*
1. Shock and hemorrhage.
2. Fat embolism.
3. Crush syndrome.
4. Pulmonary embolism.

A. Specific and Local Complications

Nonunion

When a fracture fails to show progressive signs of union at review, both clinically and radiologically, for a consecutive period of three months after the specified time expected for union, it can be defined as nonunion.

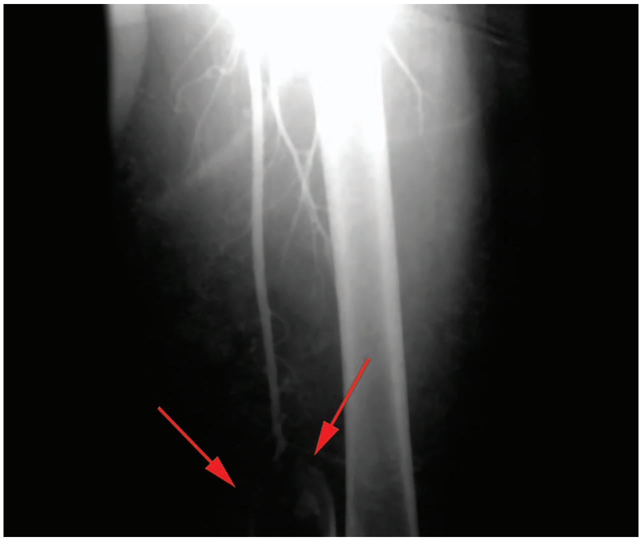


Figure 1.14

A femoral artery arteriogram showing injury to the femoral artery secondary to a badly displaced fracture of the lower third of the shaft of the femur (Vascular injury in a fracture is an emergency. Hemorrhage, shock and compartment syndrome is common. It has to be treated immediately to save the limb and the life of an individual).

Clinically, it is diagnosed by *painless abnormal mobility.* Radiologically, it shows rounding of the ends of the bones, sclerosis of the margins and poor callus. The fracture site is not viable and the *biological response of union has ceased.*

Nonunion is seen commonly in following fractures because of damage to the vessels resulting in loss of / poor blood supply.

a. Fracture of the waist of the scaphoid.
b. Fracture of the neck of the femur.
c. Fracture of the neck of the talus.
d. Fracture of the lower 1/3rd of the tibia and fibula.

Delayed Union

A fracture is said to have gone in for delayed union when there is undue delay at union.

This is so because the attempt at union for some reason does not progress towards complete union or the attempt at union is not strong enough or adequate enough to progress towards complete union.

Clinically, it is diagnosed by *painful abnormal mobility*. Radiologically, it shows callus but, the callus formed is not adequate enough to bridge the fracture site and cause complete union. The fracture site is viable and *there exists a biological response at the fracture site (but inadequate).*

Note: *Abnormal mobility is a sign to be elicited in nonunions and delayed unions. It can be elicited only in a case where implant has not been used or when implant used, has failed (loosening or breaking).*

Malunion (Figs 1.15 and 1.16)

When a fracture unites in anatomical malalignment, it is known as malunion. Possible malalignments are *angulation, rotation and over-riding.* Single or uniplanar malalignment, is rare. Usually, there is combination of two or all the three malalignments.

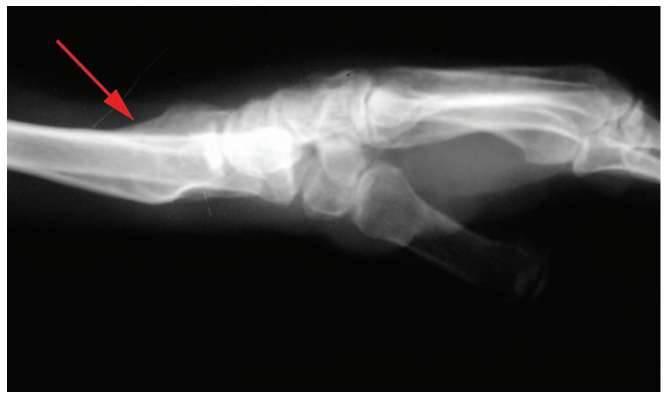


Figure 1.15

Lateral view of wrist and hand showing malunited Colles' fracture with a classical 'Dinner fork' deformity.

Figure 1.16

Malunion of a comminuted intertrochanteric fracture.
Note the neck shaft angle. It is reduced. Hence, there is coxa vara deformity at the hip.
(Left alone most of the intertrochanteric fractures unite in coxa vara. This happens because of the cancellous nature of the bone that is present at the fracture site).

Malalignment causes deformity. Angulation causes angular deformity, rotation causes rotational deformity and over-riding causes shortening.

Clinically, it is diagnosed by the presence of deformity with no abnormal mobility. Radiologically, it shows deformity and malalignment with adequate bridging callus.

> ***The normal 'Neck shaft angle' between the head, neck and shaft of the femur varies from 117-137°, average being 127°. If it is less than 117°, it is known as Coxa Vara and more than 137°, it is known as Coxa Valga. An intertrochanteric fracture malunites in Coxa Vara.***

Classification of Delayed and Nonunions (Pseudo-arthrosis)

Basically, fractures which do not unite and need a secondary procedure for achieving union are considered as the ones that have gone in for delayed union or nonunion. They show both clinical and radiological features of the same.

Depending on Strontium 85 uptake at the ends of the fracture, the vascularity (viability) is assessed and classified as hypervascular (hypertrophic) and avascular (atrophic). *(Based on Description by Weber BG and Cech O; Pseudarthrosis, Berne Switzerland 1976, Hans Huber Medical Publisher.)*

Hypervascular nonunions (true delayed unions) (Fig. 1.17A)

a. *Elephant foot type:* Presents with exuberant expansile callus and the picture resembles the foot of an elephant. It is the result of movement at the fracture site before union occurs e.g. premature weight-bearing.
b. *Horse hoof type:* Presents with little callus and picture resembles a horse hoof. Perhaps, this is the result of instability at the fracture site following inadequate reduction or fixation.
c. *Oligotrophic type:* These are hypervascular but, are not hypertrophic and do not show callus.

They are considered to be the result of major displacement/distraction persisting after treatment.

Avascular nonunions (true nonunions) (Fig. 1.17B)

a. *Torsion wedge type:* Seen when there is an intermediate fragment with poor blood supply. It unites on one side but does not unite on the other.
b. *Comminuted type:* It is the result of many intermediate fragments with poor blood supply.
c. *Defect type:* Seen when there is considerable bone loss.
d. *Atrophic type:* Seen when small intermediate fragments are missing. The defect is replaced by scar tissue.

(See the X-ray pictures—Figs 1.18A to G).

Treatment of Delayed and Nonunions

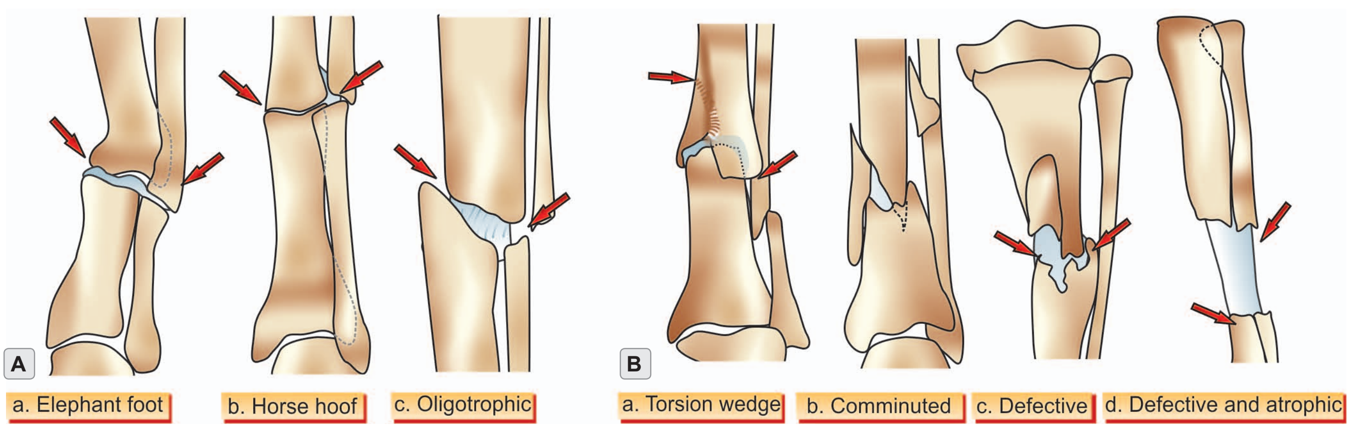
Standard Methods

Bone grafting

a. Cancellous

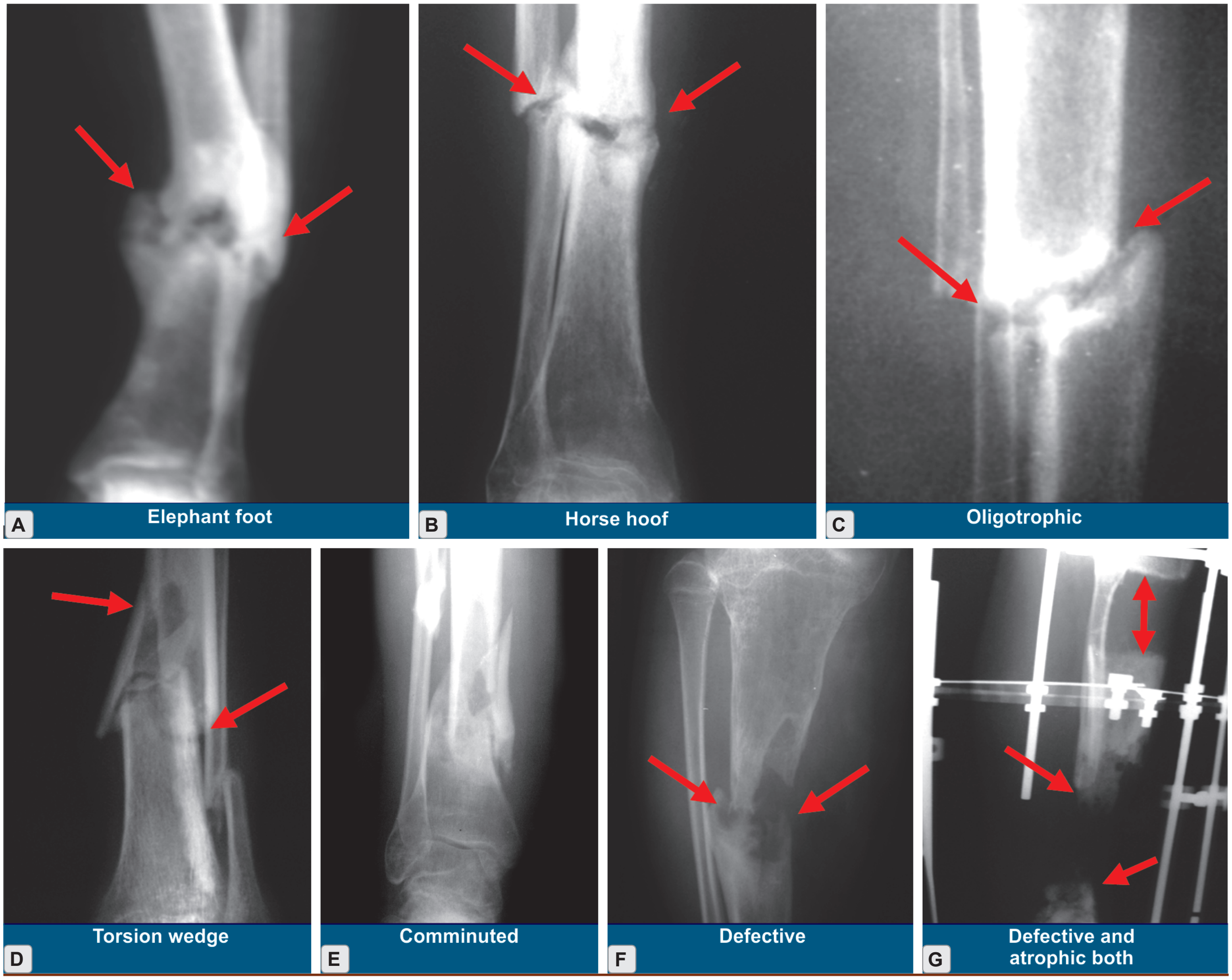
   Cancellous bone grafting is the procedure of choice to achieve union as it is osteogenic. Cancellous bone grafting is done only after confirming good apposition of the ends of bone without any soft tissue interposition. Freshening the edges of the fractured bone is a must to induce bleeding and facilitate the vascularization of the graft.
b. Cortical

   Cortical grafting is done to bridge the defect e.g. defect nonunions.



Figures 1.17A and B

Types of nonunion (Pseudo-arthrosis) (A) Hypervascular type; (B) Avascular type
(Courtesy: Based on description by Weber BG and Cech O; Pseudarthrosis, Berne Switzerland 1976, Hans Huber Medical Publisher).



Figures 1.18A to G

Different types of pseudo-arthrosis based on the description by Weber BG and Cech O. Figure 1.18G shows a bone distraction (transportation) procedure in an attempt to salvage the limb, being carried out in a combined, both defective and atrophic nonunion, employing Illizarov methodology.

In delayed union with exuberant callus and when it is certain that movement taking place at the fracture site is preventing union, rigid fixation and immobilization alone may result in union.

To conclude, the role of cancellous graft is to induce osteogenesis and the role of cortical graft is to bridge the defect. Hence, when the need is osteogenesis only, cancellous bone grafting is the procedure of choice. When the need is to bridge the defect as well as to induce osteogenesis, both cortical and cancellous bone grafting (cortico-cancellous grafting) is the procedure of choice.

The procedure needs immobility at the site for the incorporation of the graft and the union to take place. Thus, internal fixation/external immobilization is necessary.

Sources of bone graft

a. Autograft—from the same individual.
b. Allo/Homograft—from the same species, e.g. bone bank, maternal fibula.
c. Isograft—from an identical twin.

Source of cancellous graft: Iliac crest, excised ribs, excised head and neck of femur.

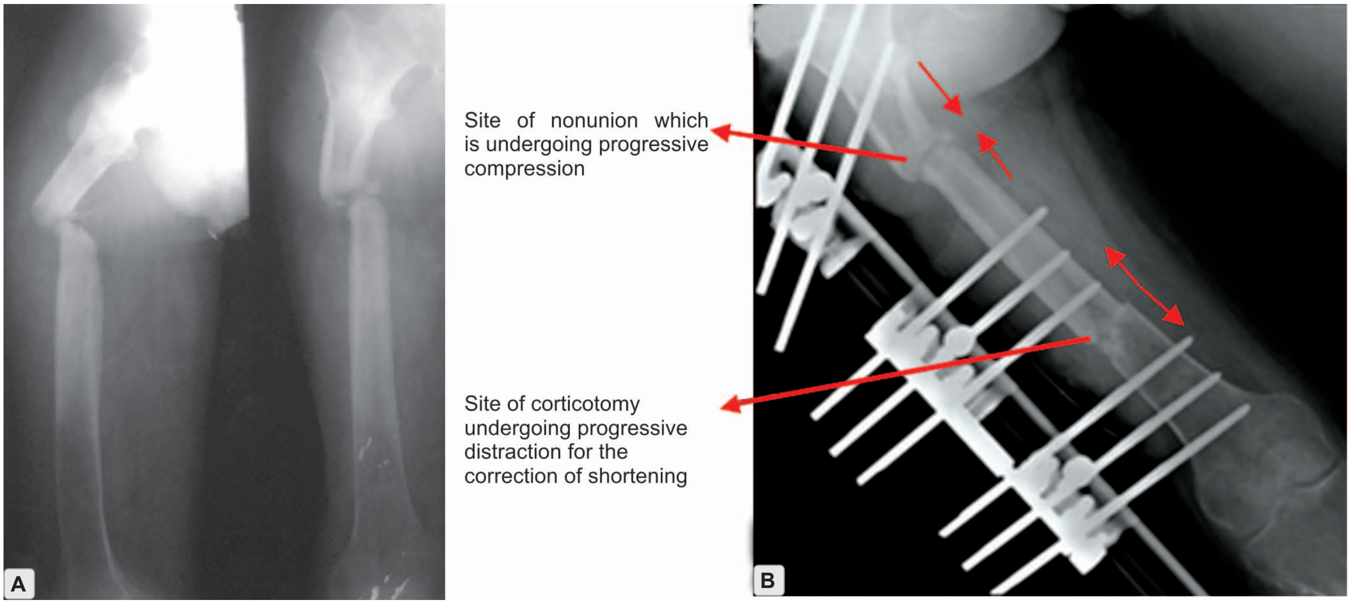
Source of cortical graft: Upper 2/3rd of the fibula, anteromedial tibia.

Specialized Methods for the Treatment of Delayed Unions and Nonunions

a. Distraction/compression osteogenesis based on the principle of Ilizarov: It has been proved by Ilizarov that controlled progressive distraction and/or compression leads to tissue regeneration. Procedures based on this principle are of immense use when the skin condition does not permit open bone grafting procedures (Figs 1.19A and B).

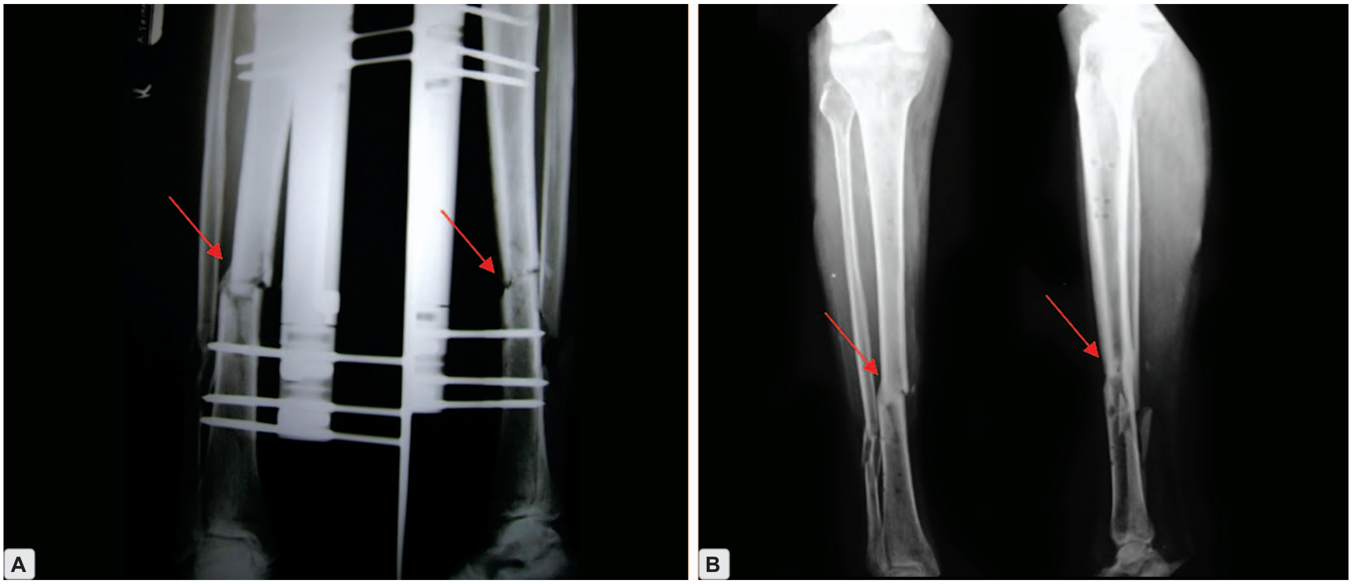
b. Bone marrow grafting procedure: In this, bone marrow is aspirated and injected into the site of nonunion/delayed union. The aspirate may be injected as such before clotting or may be injected in a concentrated form with anticoagulants, obtained after centrifuging under aseptic precautions (Figs 1.20A and B).

c. Implantation of bone morphogenic protein along with collagen sponge (bovine type-I) has been



Figures 1.19A and B

(A) Nonunion of fracture shaft femur associated with shortening of the limb; (B) Procedure of compression/distraction osteogenesis.



Figures 1.20A and B

Union in a 12 weeks old ununited type III open fracture of tibia and fibula after 2 injections of bone marrow grafting at 6 weekly intervals.

found to be useful in the treatment of delayed union and nonunion of long bones. Recombinant human proteins such as rhBMP-2 and osteogenic protein OP-1 are available for commercial use. The osteogenic protein comes in powder form which is mixed with collagen in sterile saline solution to form a paste. This paste is then injected at the site of nonunion. The preparation induces bone formation by stimulating mesenchymal cells to differentiate into chondroblasts and osteoblasts.

Treatment of Malunions

Corrective osteotomy: In this procedure, the site of malunion is osteotomized surgically and the deformity is corrected accordingly. The alignment thus obtained, is maintained by internal fixation devices and appropriate external immobilization (very rarely by external cast alone). Immobilization is continued till healing takes place.

Osteoclasis: This is a closed procedure wherein the malunited bone is broken manually, reduced, aligned and immobilized in an appropriate cast. This procedure is indicated in early cases of malunion in children where the soft bridging callus has just formed. Osteoclasis should never be attempted after consolidation of callus.

The summary of different methods of fracture treatment is shown in Flow chart 1.1.

Sudeck's Atrophy (Figs 1.21A and B)

Synonyms are Sudeck's osteodystrophy, reflex sympathetic dystrophy, complex regional pain syndrome, causalgia, shoulder hand syndrome.

It is a chronic condition characterized by pain and stiffness in the extremity as a result of dysfunction of central or peripheral nervous system.

Pathogenesis is thought to be due to:

a. Activation of pain pathways by the release of catecholamines, e.g. norepinephrine following injury.
b. Exaggerated inflammation and immune response following injury.

Signs and symptoms

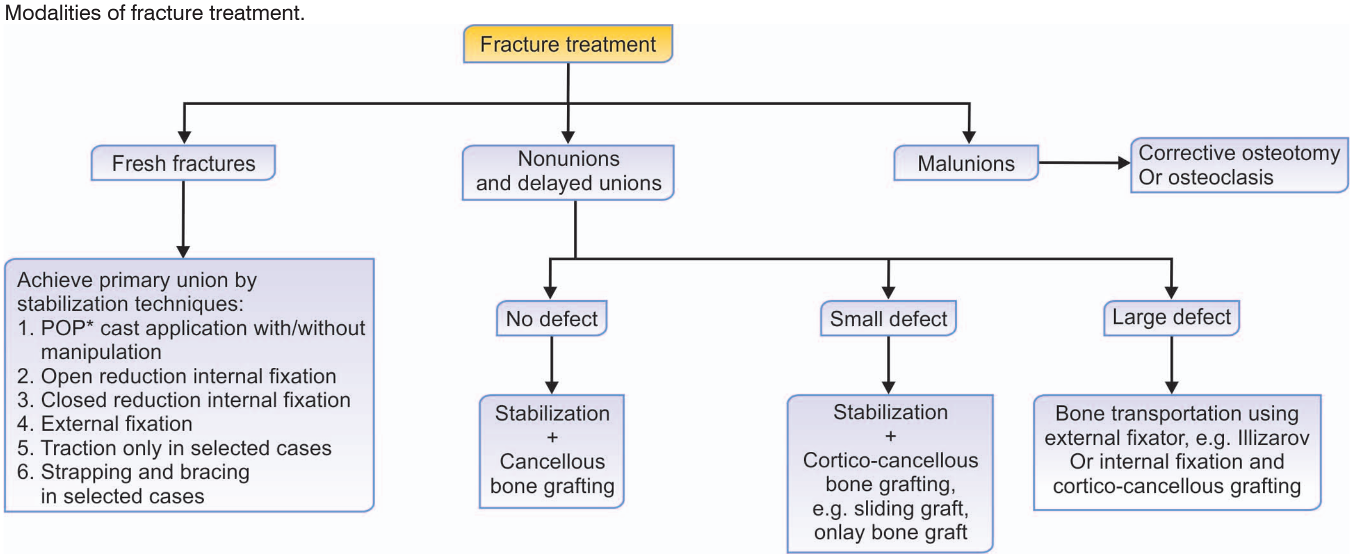
a. Burning pain.
b. Skin temperature changes (warm initially, cold later).
c. Skin color and texture changes.
d. Alteration in nail and hair growth.
e. Swelling and stiffness of joints.
f. Impairment of function of extremity.

Diagnosis

Diagnosis is made by clinical signs, X-ray (shows demineralization) and nuclear bone scan (shows increased uptake).

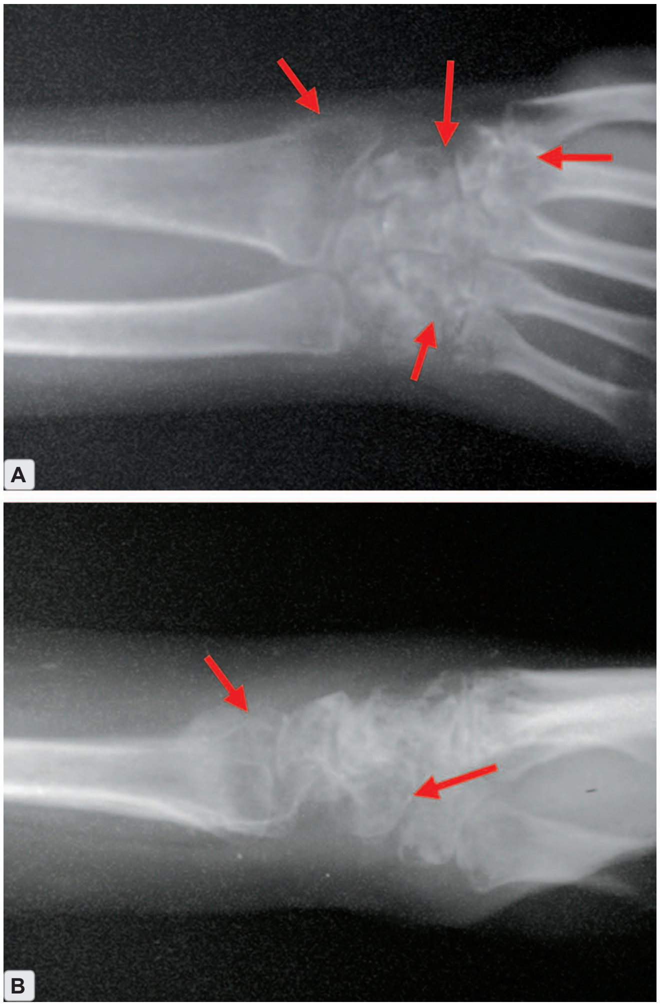
Flow chart 1.1

Modalities of fracture treatment.



*POP—Plaster of Paris

Note: *Infection when present should always be adequately treated before surgical procedures are carried out in fresh fractures as well as in nonunions and delayed unions.*



Figures 1.21A and B

A normally united Colles' fracture with Sudeck's osteodystrophy. Note the severe nature of demineralization and rarefaction in the cancellous areas of the bones of forearm wrist and hand.

Treatment

Treatment aims at reducing sympathetic overactivity.

Drug therapy is given using following drugs as per need:

- Opiates
- Anti-inflammatory analgesics
- Antidepressants
- Antiepileptics
- Calcitonin
- Corticosteroids

Interventional therapy as follows:

- Physical and exercise therapy
- Psychotherapy to relieve anxiety and depression
- Sympathetic nerve block
- Spinal cord stimulation
- Intrathecal drug pump

B. Systemic and General Complications

Shock and Hemorrhage

Hemorrhagic shock: This is the most common complication of a severe injury. Inadequate oxygenation of tissues leads to a chain of events resulting in multiorgan failure and death.

Measures to be adopted include:

a. Adequate oxygenation of tissues by maintaining the airway and administration of oxygen (important and to be employed in all types of shock).
b. Replacement of lost volume by infusing saline/ Ringer's lactate and blood.
c. Immediate stoppage of bleeding.

Diagnosis of hemorrhagic shock is made by cold and clammy feeling of the body, hypotension (initially diastolic and later both systolic as well as diastolic), tachycardia, tachypnea and poor urine output. Loss of more than 40% of the blood volume will pose grave danger to life.

Neurogenic shock: This type of shock is commonly seen in spinal cord injury. The disturbance in sympathetic innervation causes decrease in the heart rate, dilatation of peripheral vessels and as a result fall in blood pressure. Monitoring the central venous pressure and infusion of plasma expanders help in the management of this difficult problem.

Cardiogenic shock: In a trauma setting, this type of shock commonly results from chest injuries, e.g. tension pneumothorax impeding venous return, myocardial contusion, etc. should be managed promptly depending on the underlying cause.

Septic shock: This type of shock is seen as a result of septicemia and usually occurs several days after the open injury as a result of septicemia.

It is characterized by increased warmth of the body, tachycardia, hypotension (little fall in systolic but marked fall in diastolic pressure) and tachypnea. Multiorgan failure is common. Septicemia with gram-negative organism may not increase the body warmth.

Aggressive antibiotic therapy along with all other supportive measures is essential for the recovery.

Fat Embolism

In long bone fractures, there is always dissemination of fat globules from the marrow into the bloodstream. This can also happen from a spongy bone. At times, these fat globules block the capillaries of the pulmonary and the cerebral vessels causing fat embolism syndrome. The syndrome is more common in a young patient with multiple fractures.

Diagnosis of the fat embolism is difficult. But the following features developing after a fracture should arouse suspicion.

a. Confusion and restlessness
b. Increased body temperature and tachycardia in a patient who is otherwise normal and not in a state of shock
c. Breathlessness
d. Petechiae over the chest, back, axilla, the conjunctival folds and the retina

In severe cases of pulmonary embolism, there can be blood tinged frothy secretion which is coughed out by the patient. In case of cerebral embolism, patient may become comatose. There is no definite treatment for fat embolism except supportive measures of giving high concentration of oxygen and maintaining capillary perfusion. Low molecular weight dextran may help in maintaining capillary perfusion. If the oxygen saturation falls severely, intubation and ventilation is the treatment of choice. If there is blood loss and hemoglobin is low, blood has to be transfused. Good quality blood is essential for maintaining the oxygen saturation.

Crush Syndrome

First described by a British physician Eric Bywaters in the year 1914. It is a traumatic rhabdomyolysis occurring because of crushing. Also known as reperfusion injury, it occurs in those whose limbs are compressed for a long-time, e.g. limb trapped in a vehicular collision or buried in a land slide, etc. Such a limb is deprived of blood flow for quite a long period and the tissues release toxic metabolites because of cell death. When the compression is released and reperfusion occurs, myoglobin from the dead muscle and the toxic metabolites enter the bloodstream.

The flow of oxygenated blood through the damaged area causes formation of reactive oxygen metabolites resulting in further damage to the tissues. The resultant hyperkalemia, hypocalcemia and metabolic acidosis may cause cardiac arrest. Renal failure may occur when myoglobinuria is severe and blocks the renal tubules.

Management: Large amount of fluid infusion to dissolve the metabolites and simultaneous forced diuresis with the help of diuretics is the treatment of choice till myoglobinuria becomes negligible. Crushed tissues should be radically debrided and a tight compartment when present should be adequately decompressed. Dialysis may be necessary when renal failure is observed.

Pulmonary Embolism

Pulmonary embolism commonly occurs in those patients who are confined to bed for a prolonged period after fracture. Elderly high-risk patients who present with major fractures are more prone to develop pulmonary embolism. Prevention by heparinization of blood with the help of low molecular weight heparin is the best measure employed in such patients.

Recent Concept of Fracture Healing

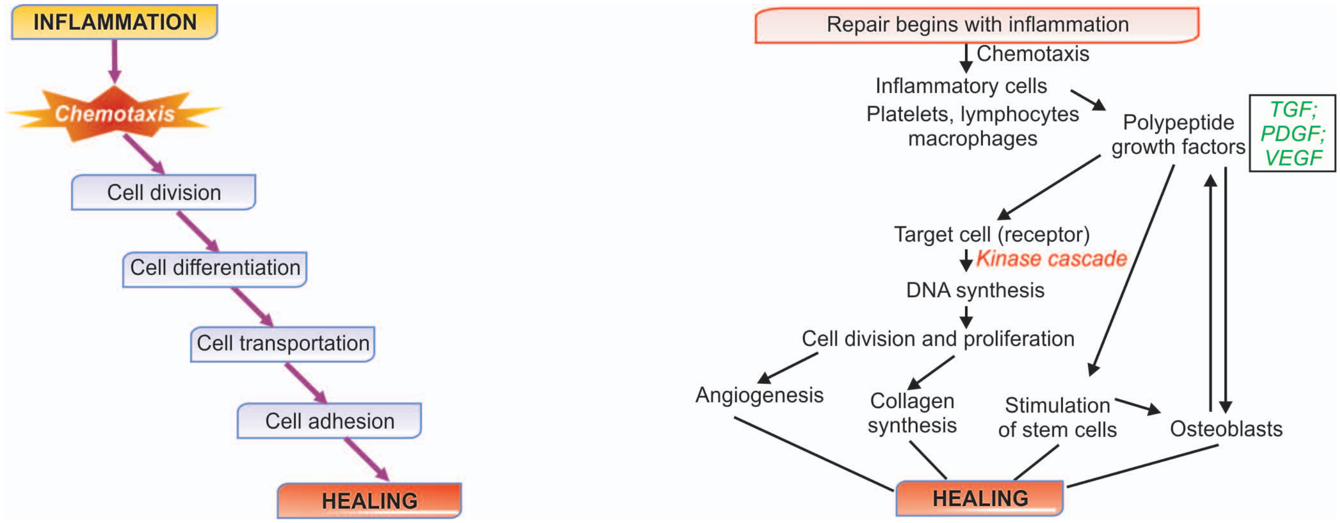
Regenerative Medicine

Regenerative medicine is an evolving branch which tries to restore the normalcy from a diseased state, by analyzing the cellular and molecular responses. Thus, if fracture healing is analyzed, it is found that the healing process begins with inflammation. A series of events following inflammation, deliver to the site of fracture several chemical mediators, cells and growth factors (TGF), etc. This in turn initiates a chain of reactions which results in fracture healing (Flow chart 1.2).

Thus, after understanding the exact process of healing, these days, methods which are different from conventional ones have evolved. Bone marrow grafting, Stem cell injection alone or insertion of a scaffold mixed with stem cells, bone morphogenic proteins (rhBMP-2.OP-1) along with collagen are some of the methods currently practised. In the years to come, this concept is going to revolutionize the treatment of fractures and nonunions.

Flow chart 1.2

Schematic representation of the chain of events occurring at the cellular level which is responsible for the healing of a fracture.



DISLOCATIONS

Definition of Dislocation and Subluxation

When the two articular surfaces are totally out of contact, it is known as dislocation.

(Total loss of contact between the two articular surfaces).

When the two articular surfaces are partly in contact and partly out of contact, it is known as subluxation. Subluxation is also known as partial dislocation.

(Partial loss of contact between the two articular surfaces).

Types of Dislocation (Figs 1.22A to D)

a. Traumatic
b. Pathological, e.g. pyogenic arthritis
c. Paralytic, e.g. poliomyelitis
d. Congenital, e.g. DDH, congenital dislocation of the knee

Note: *In a dislocation, the part which loses contact with the rest of the body is considered as minor segment. The position which this part, i.e. the minor segment occupies in relation to the major segment classifies the dislocation as anterior, posterior, medial, lateral and central.*

When there is a fracture involving the articular surface of a joint or the adjoining bone, along with dislocation the term fracture dislocation is used to describe the injury (Figs 1.23A and B).

Traumatic Dislocation

For a traumatic dislocation to occur, the force should be of a considerable magnitude.

Diagnosis

Clinical diagnosis is simple because of the classical attitude and signs. Total loss of joint movement is characteristic and any attempted movement results in severe pain.

Treatment

Any dislocated joint has to be reduced under anesthesia as early as possible. Delay poses difficulty in reduction. This is because of re-organization of muscles and soft tissue structures around the joint. Irreducibility under anesthesia with adequate muscle relaxation, indicates entanglement of the dislocated part in the surrounding tissues such as joint capsule, muscles and tendons and rarely vessels and nerves.

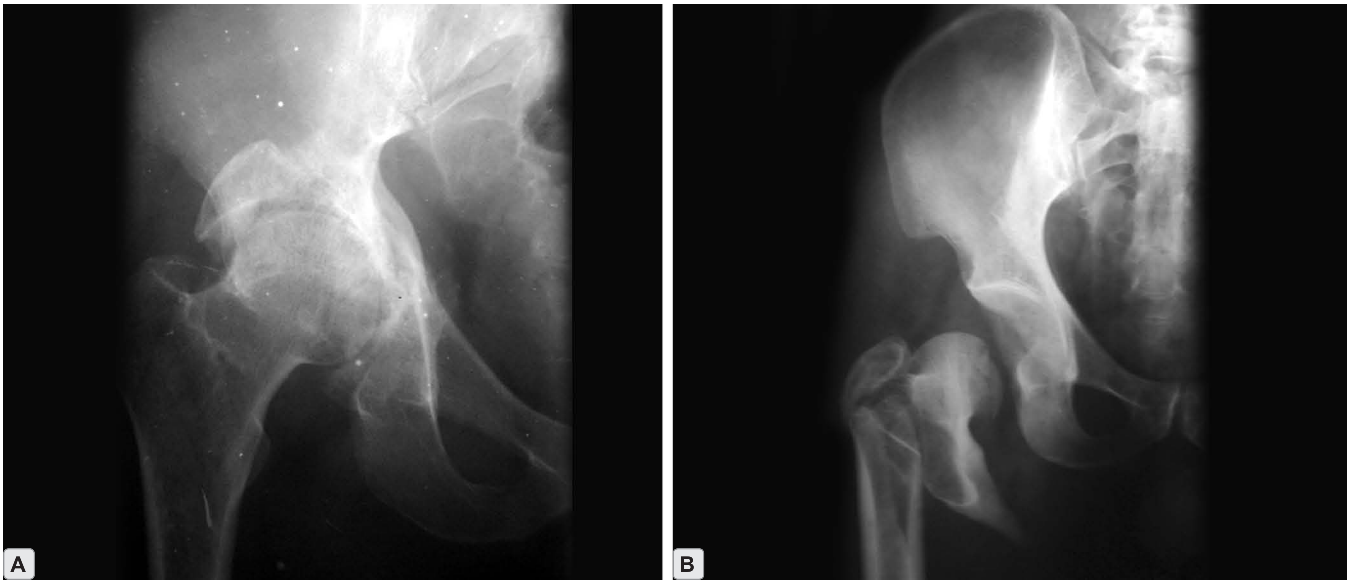
Following successful reduction, immobilization for a minimum period of three weeks is essential. This is necessary for healing of damaged soft tissues. Poor healing of damaged soft tissues increases the chance of recurrent dislocation and subluxation.

Complications of Dislocation

i. Injury to the neighboring nerves and vessels, e.g. sciatic nerve injury in posterior dislocation of the hip, popliteal artery in posterior dislocation of the knee, etc.

Figures 1.22A to D

Different types of dislocations. (A) Traumatic anterior dislocation of the hip. (B) Pathological dislocation—secondary to tuberculosis of the hip joint. (C) Paralytic dislocation—secondary to post polio residual paralysis. Note a recent fracture in the thin femur. (D) Neglected developmental dysplasia of the hip.



Figures 1.23A and B

Anteroposterior view of the hip joint showing: (A) Fracture of the acetabulum with posterior subluxation of the hip joint. (B) Comminuted Intertrochanteric fracture with posterior dislocation of the hip joint. Both are fracture dislocations.

ii. Recurrent dislocation.
iii. Habitual dislocation.
iv. Avascular necrosis.

Pathological Dislocation

Pathological dislocation is a type of dislocation where the dislocation is the result of a pre-existing pathology. In this, it is important to ascertain whether the primary pathology that was responsible for dislocation, is still active. If it is active and persistent, e.g. persistent infection in a pathological dislocation due to infective arthritis, control of infection is achieved first before the dislocation is treated. The secondary changes that have occurred as a result of primary insult are treated by means of reconstructive procedures. These reconstructive procedures vary with respect to joint and age. They are categorized as joint replacement procedures, osteotomies and arthrodesis.

Paralytic Dislocation

In this, the dislocation occurs due to muscle imbalance secondary to muscle paralysis. Whenever possible, the paralyzed muscles are substituted with normal muscles by carrying out procedures such as muscle and tendon transfers. When not possible, e.g. in cases of long standing paralytic dislocation, procedures like osteotomy and arthrodesis are considered.

Revision Questions

Q. Define a fracture. How do you classify fractures?
Q. Describe the different stages of fracture healing.
Q. Define and classify nonunions. Discuss the management.
Q. What are the factors responsible for delayed union and nonunion? Discuss the management of delayed and nonunion.
Q. Enumerate the complications of a fracture. Discuss their management.

Note: For questions on dislocations refer Chapter 4.

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2 Fractures in the Upper Limb

General information, mechanism of injury, diagnosis, treatment and complications are dealt with in the following order:

- *Fractures around the wrist*
- *Fractures around the elbow*
- *Fractures in the forearm*
- *Fractures in the arm and around the shoulder*

FRACTURES AROUND THE WRIST

The common fractures that occur around the wrist joint are:

1. Bennett's fracture.
2. Rolando's fracture.
3. Scaphoid fracture.
4. Colles' fracture.
5. Smith's fracture.
6. Barton's fracture.
7. Essex-Lopresti fracture.
8. Chauffeur's fracture.

These are eponyms (except scaphoid fracture) and are named after the person who described the fracture.

Bennett's and Rolando's Fracture

General Information
Bennett's and Rolando's fractures are intra-articular fractures involving the base of the thumb metacarpal. Restoration of articular surface congruity is of utmost importance while treating these fractures. Persistent articular surface incongruity results in painful limitation of movements and later, carpometacarpal degenerative arthritis. This may require treatment in the form of arthrodesis or arthroplasty for restoring useful function of the thumb.

Bennett's Fracture

Definition

It is an oblique fracture occurring at the base of the 1st metacarpal bone (thumb metacarpal) with subluxation of the carpometacarpal joint.

Who Described this Fracture?

It was described by Edward Hallaran Bennett, MD, an Irish surgeon in the year 1882 and hence known as Bennett's fracture.

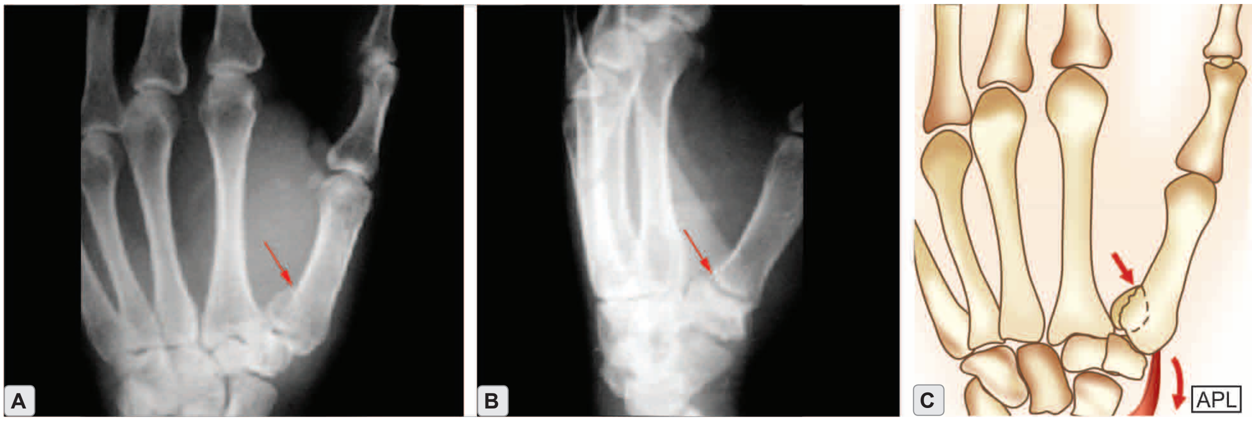
Note: *He suggested that early diagnosis and treatment of this fracture is absolutely necessary to prevent complications of highly mobile carpometacarpal joint of the thumb.*

Mechanism of Injury

The mechanism of injury is an axial loading of partially flexed thumb metacarpal, e.g. delivery of a punch with a clenched fist.

Displacements

The two fragments are placed as follows after the fracture (Figs 2.1A to C).



Figures 2.1A to C

(A and B) Radiographs of Bennett's fracture showing classical displacements; (C) Line diagram of the same showing the fracture and the displacement occurring due to the pull of abductor pollicis longus (APL) tendon.

The ulnar fragment remains in place and the radial fragment is displaced.

- *The volar ulnar triangular fragment*—is held firmly in place by the volar oblique ligament.
- *The radial distal fragment*—is displaced radially, dorsally and proximally by the pull of the abductor pollicis longus tendon.

Treatment

A. *Nonoperative:* Fractures with < 1–2 mm disruption of articular surface and with minimal displacement are treated by closed manipulation, reduction and maintaining the reduction in thumb spica cast for a period of 6–8 weeks.

B. *Operative:* Closed reduction/Open reduction and K-wire fixation and immobilization in thumb spica for a period of 6–8 weeks.

Gradual mobilization is to be commenced after removal of spica cast.

Note: *"Kirschner wire" also known as K-wire in short is a thin stainless steel wire with a sharp-pointed tip designed by Martin Kirschner, 1909.*

Complications

1. Osteoarthritis of carpometacarpal joint (due to joint incongruity and articular surface injury).
2. Stiffness and loss of mobility of carpometacarpal joint of the thumb (result of prolonged immobilization).
3. Loss of reduction and recurrent subluxation with instability.
4. Surgical complications of infection and injury to sensory branch of the radial nerve.

Revision Questions

Q. Define Bennett's fracture.
Q. Who described Bennett's fracture?
Q. What is the mechanism of injury?
Q. What are the displacements?
Q. Describe the management of Bennett's fracture.
Q. What are the complications of Bennett's fracture?

Essay Question

Q. Define Bennett's fracture. Discuss the mechanism of injury, diagnosis and management of Bennett's fracture. Enumerate its complications.

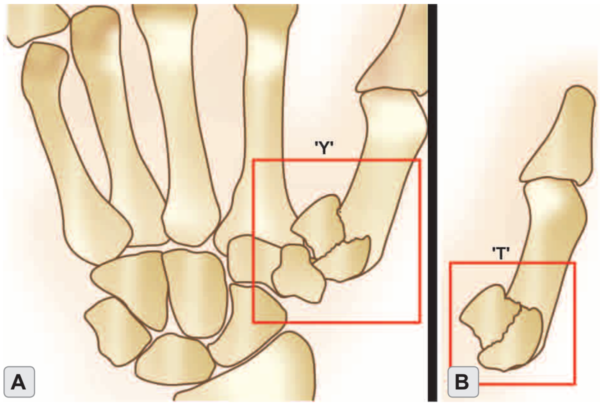
Rolando's Fracture

Definition

It is a three part fracture involving the base of the thumb metacarpal with intra-articular extension and subluxation of carpometacarpal joint. The fracture line is in the shape of 'T' or 'Y' (Figs 2.2A and B).

Who Described this Fracture?

It was described by Silvio Rolando in the year 1910 and hence known as Rolando's fracture.



Figures 2.2A and B
(A) 'Y' type of Rolando's fracture; (B) 'T' type of Rolando's fracture.

Mechanism of Injury

It is a result of axial loading during abduction of the thumb. For example, impaction during a sporting event, impaction of a steering wheel or a handle bar.

Treatment

A. Badly comminuted fractures are treated in thumb spica immobilization for 3 weeks followed by gradual mobilization.
B. Three part fractures are treated by open reduction and internal fixation using K-wires or mini T plates and screws and immobilization in thumb spica. Later, after 2–3 weeks gradual mobilization is started depending on the stability of fixation.

Prognosis

Prognosis is generally poor. Carpometacarpal degenerative arthritis is common which needs arthroplasty or arthrodesis.

Revision Questions

Q. Define Rolando's fracture.
Q. Why is it known as "Rolando's fracture"?
Q. What is the mechanism of injury?
Q. How is this fracture treated?
Q. What is the prognosis in Rolando's fracture?

Scaphoid Fracture

General Information

The word scaphoid is derived from Greek language. "Skaphos" in Greek means boat.

Scaphoid is a boat shaped bone. Scaphoid bone is also known as navicular. The articular cartilage covers 80% of the bone surface. It is placed at 45° to the longitudinal axis of the wrist. It articulates with capitate, trapezium, and trapezoid distally, distal radius proximally and lunate medially.

Fracture scaphoid accounts for 60% of carpal injuries. Commonly seen in young adults. Rare in children and elderly. Because of relative weakness of lower radius, in elderly, a fall on an outstretched hand results in fracture of the lower radius and not the scaphoid.

Unlike other fractures, it does not produce severe symptoms. Hence, there is a chance of delay in diagnosis. This delay when significant can result in a variety of adverse outcomes such as delayed union, nonunion, avascular necrosis, decrease in grip strength, limitation of joint movements and radiocarpal degenerative arthritis. So, timely diagnosis and appropriate treatment is absolutely essential to prevent these complications.

Blood Supply of Scaphoid

The bone scaphoid derives its blood supply from the branches arising from superficial branch of radial artery. They enter the bone distally and then run proximally (Fig. 2.3).

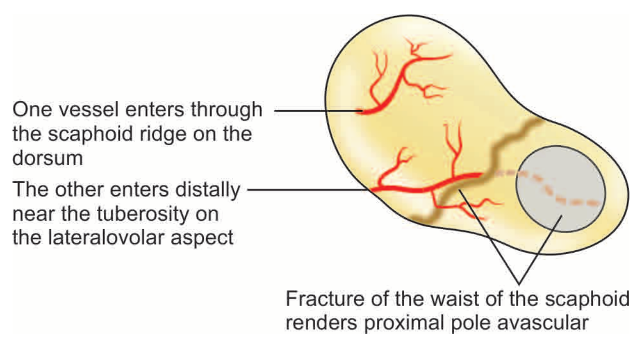
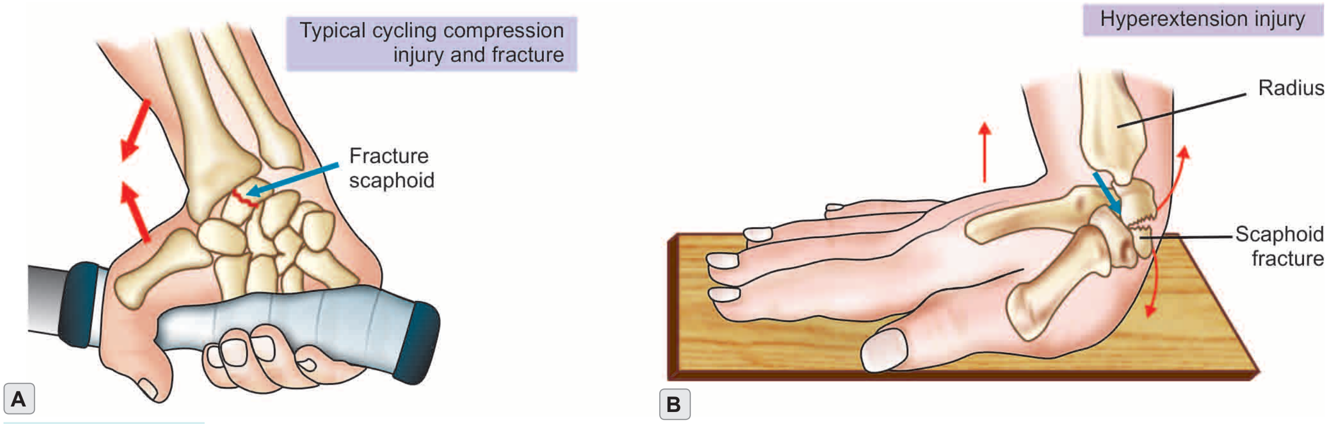


Figure 2.3
Blood supply to the scaphoid (Right : PA view). Note that the vessels enter the bone distally and runs proximally. These are branches arising from superficial branch of radial artery.



Figures 2.4A and B

Pictures show typical (A) Radial compression and (B) Hyperextension injuries.

Mechanisms of Injury (Figs 2.4A and B)

Two mechanisms are described.

1. Radial compression and dorsiflexion occurring at the wrist during a fall on an outstretched hand: generally results in an undisplaced fracture.
2. Hyperextension occurring at the wrist during a fall on an outstretched hand: generally results in displaced fracture scaphoid.

Undisplaced fractures are less complicated than displaced fractures.

Classifications

A. Based on Mayo classification (Fig. 2.5):
   1. Fracture of the tuberosity
   2. Fracture of the distal body
   3. Fracture of the waist
   4. Fracture of the proximal pole
   5. Osteochondral fracture

B. Based on Russe classification, the fracture scaphoid is classified as:
   i. Stable
   ii. Unstable

All displaced scaphoid fractures irrespective of the nature of the fracture line are considered as unstable scaphoid fractures.

Horizontal oblique and transverse fractures are considered as stable fractures when undisplaced (Figs 2.6A to C).

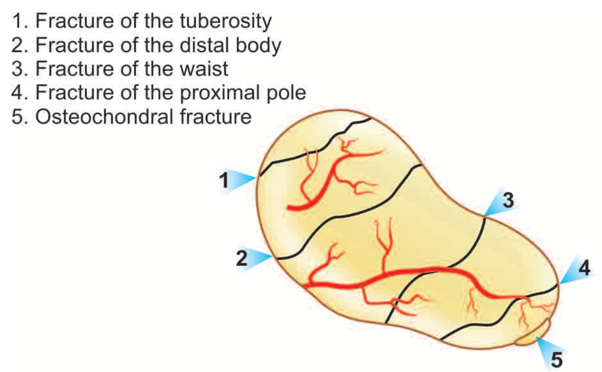


Figure 2.5

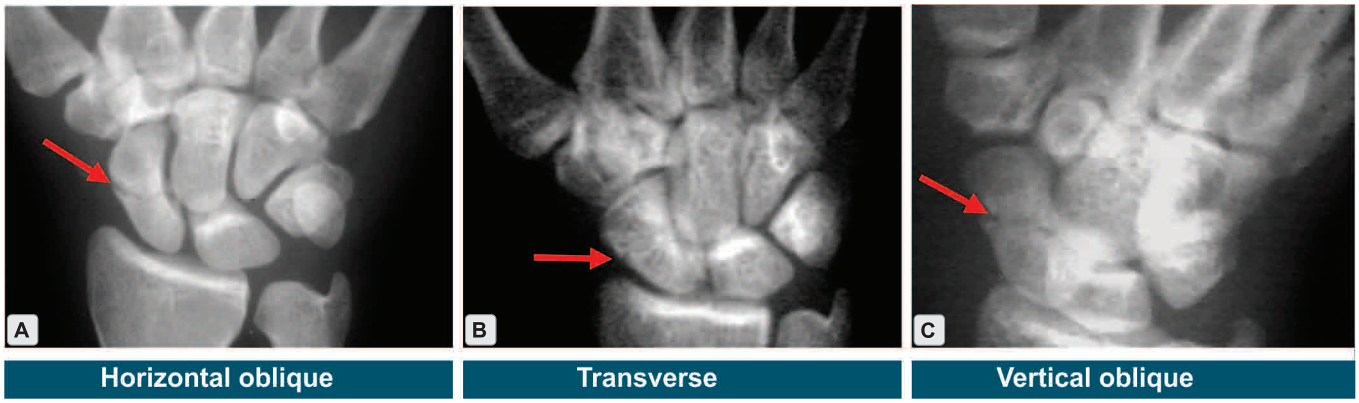
Different sites of fracture based on Mayo classification. Osteochondral fracture is difficult to identify radiologically and may need an MRI.

Clinical signs that help in diagnosis (Figs 2.7A to D)

1. Tenderness in the anatomical snuff box.
2. Tenderness over the scaphoid tubercle (Freeland, 1989).
3. Scaphoid compression test (Chen, 1989).
4. Painful limitation of movements of wrist and thumb.

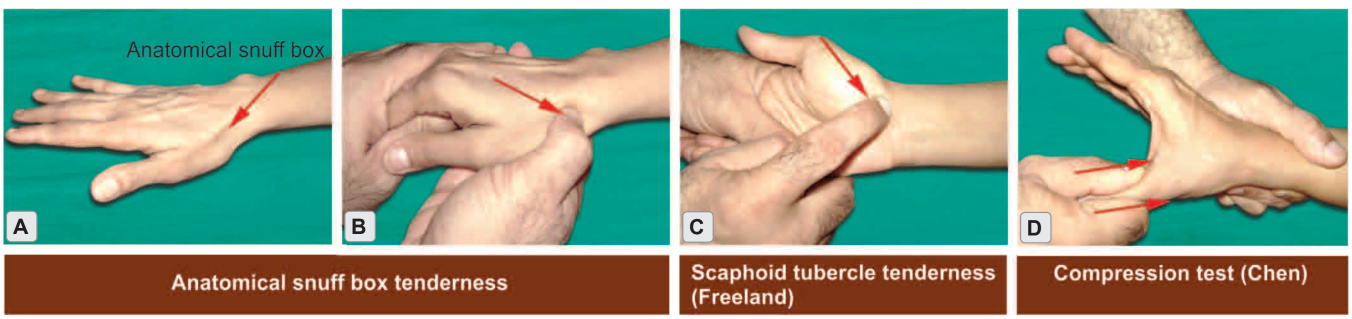
Investigations

Ninety percent of the fractures are diagnosed by the standard views for the scaphoid. Those which are not seen radiologically are either incomplete or undisplaced fractures and are likely to be diagnosed by repeat X-rays after 15 days.



Figures 2.6A to C

Radiographs of scaphoid fracture showing nature of fracture lines. Horizontal oblique and transverse fractures are considered as stable fractures when undisplaced. Whereas vertical fractures whether displaced or undisplaced are unstable.



Figures 2.7A to D

(A and B) Anatomical snuff box tenderness; (C) Scaphoid tubercle tenderness, Freeland; (D) Compression test, Chen, Note: Tenderness varies depending on the site and type of the fracture. Hence, it is advisable to make use of all the above signs in combination for a proper diagnosis.

Magnetic resonance imaging (MRI) is indicated when in doubt after the repeat X-rays. It is also done to assess the extent of avascularity and carpal malalignment as a consequence of avascular necrosis and nonunion.

Radiological views taken to diagnose fracture scaphoid

1. Standard PA view of the wrist (with wrist in dorsiflexion and ulnar deviation after making a fist)
2. Standard lateral view.
3. Radioulnar oblique view in midprone position.
4. Stress views only if needed (Stress view opens up the fracture site).

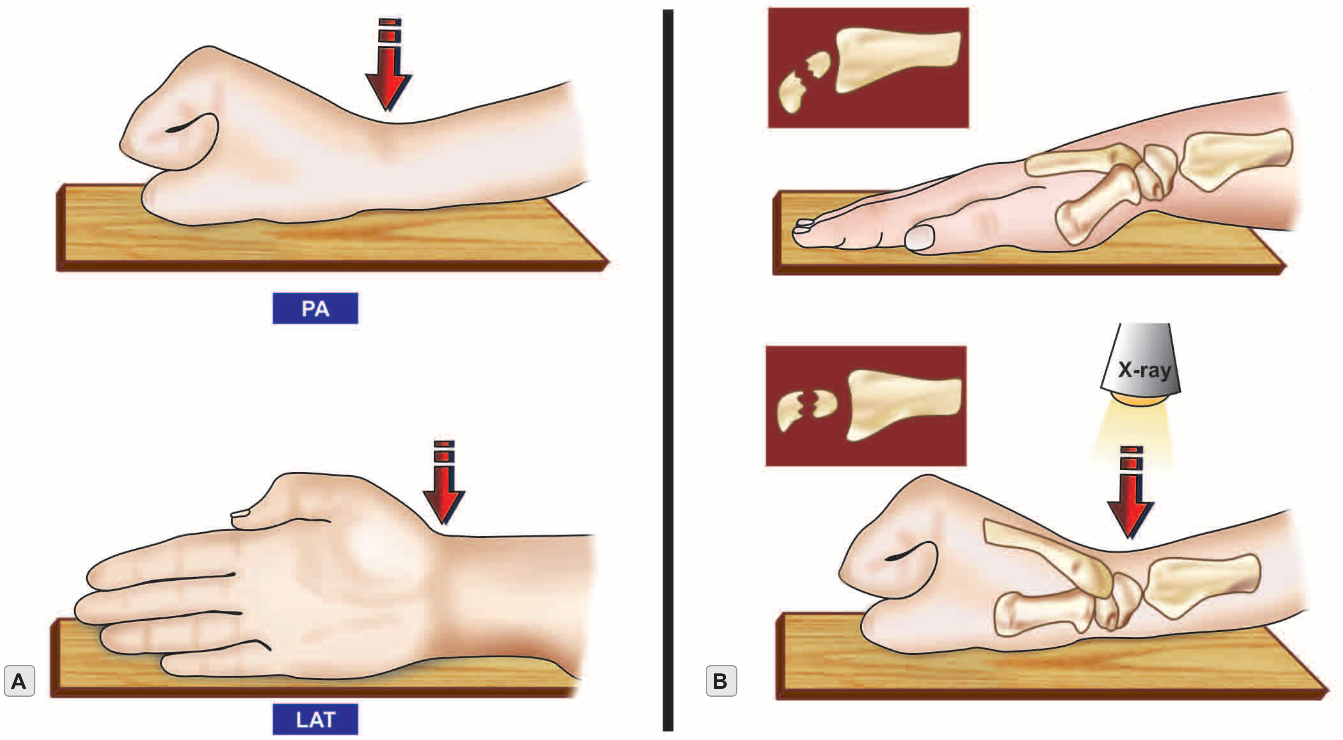
Note: *Normally scaphoid is placed at an angle of 45° volar to the longitudinal axis of the radius. Thus making a fist in PA view, brings it in alignment with the radius and parallel to the X-ray plate (Figs 2.8A and B).*

Interpretation of the radiograph: Scaphoid fracture is generally identified as (Figs 2.9 and 2.10):

a. A clear lucent line across the bone.
b. A distinct break in continuity.
c. A distinct sharp step.

Methods of Treating Fracture Scaphoid

A. *Nonoperative:* It is employed in undisplaced and incomplete fractures. A special cast known as ***Scaphoid Cast*** with wrist in 15° of dorsiflexion and 5° of radial deviation is given for 4–6 weeks. The position of immobilization is sometimes described as glass holding position (Some studies show that a standard below elbow cast is sufficient to immobilize these fractures and scaphoid cast is unnecessary).



Figures 2.8A and B
(A) PA (Posteroanterior) and lateral view; (B) Normal volar tilt of the scaphoid is negated by dorsiflexion of the wrist.

Figures 2.9A to C
(A) A thin lucent line; (B) Distinct break in a cortical continuity; (C) A sharp step.

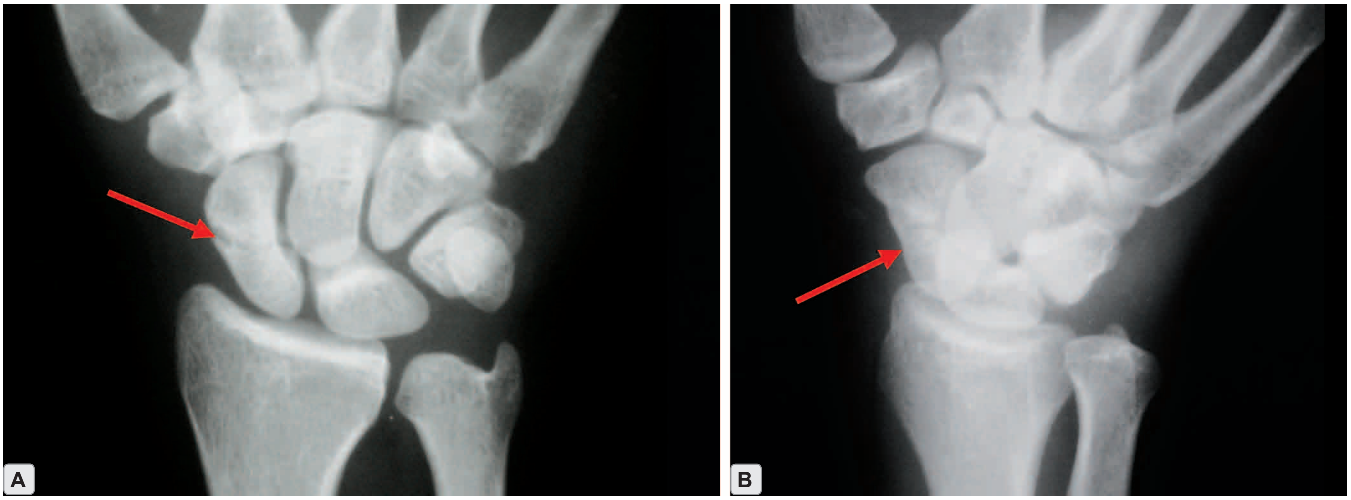
Need for immobilization in 15° of dorsiflexion and 5° radial deviation: This is because of the mechanism of injury which is radial compression and dorsiflexion. Hence, immobilization in a position of dorsiflexion and radial deviation relaxes the fracture site (Avoids tension stress leading to opening up of the fracture site).

B. *Operative:* Is employed in displaced fractures. Closed/open reduction and internal fixation done using special screws, e.g. Herbert screws, Acutrak, AO scaphoid screws, etc. This helps in early mobilization and functional recovery.

Complications of Fracture Scaphoid

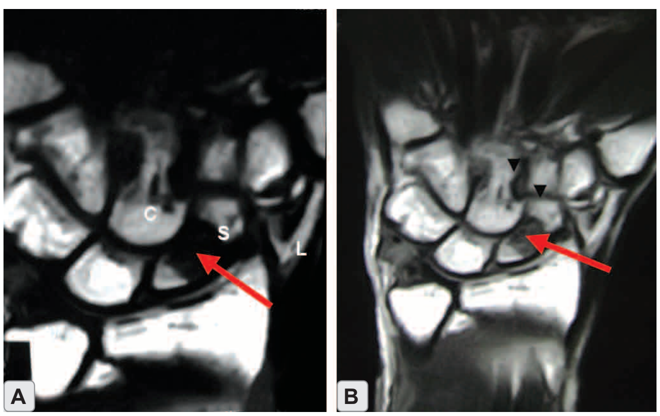
1. Nonunion
2. Avascular necrosis (Figs 2.11A and B)
3. Radiocarpal degenerative arthritis (Figs 2.12A to C)

Cause for avascular necrosis: Avascular necrosis occurs because of peculiarity in blood supply which is from distal to proximal (see Fig. 2.3). It depends on the type of fracture and the degree of displacement which again is directly proportional to the nature and severity of the injury.



Figures 2.10A and B

(A) Standard PA view; (B) Standard radioulnar oblique view. Fracture of the scaphoid is seen here as (A) a distinct break in cortical continuity, (B) A sharp radiolucent line.



Figures 2.11A and B

Nonunion of fracture scaphoid and avascular necrosis. Note the lack of density at the avascular portion of the scaphoid observed in both T1 and T2-weighted images.

Figures 2.12A to C

Scaphoid nonunion with avascular necrosis of proximal pole which has shrunk getting resorbed. This happens because of repetitive stress on the avascular bone over a long period of time. Also note the evidence of radiocarpal arthritis which is shown by narrowing of the joint space and osteophyte formation.

Treatment of the Complications of Fracture Scaphoid

Nonunion with avascular necrosis is treated by means of internal fixation and bone grafting. Several types of bone grafting techniques are described (including vascularized bone graft).

Radiocarpal degenerative arthritis is treated by wrist arthrodesis in 15° of dorsiflexion and 5–10° of ulnar deviation.

Other procedures

1. Excision of avascular proximal pole
2. Excision of the whole of scaphoid
3. Excision of proximal row of carpus
4. Replacement of the scaphoid
5. Wrist arthroplasty

Reasons why these fractures are commonly missed

1. Mistaken for minor injury or sprain by the patients because they do not cause severe symptoms.
2. Improper X-rays.
3. Incomplete and undisplaced fractures are difficult to diagnose by X-rays.

Note: *Incomplete and undisplaced fractures generally do not give rise to complications.*

Treatment of Suspected Cases of Fracture Scaphoid

When suspected, the injury should always be immobilized in a scaphoid cast/below elbow cast for a period of 2 weeks. Cast is removed at the end of 2 weeks and patient is re-examined. If signs are persistent a repeat X-ray/MRI is done for confirmation and treated accordingly.

Revision Questions

Q. What is the other name for scaphoid?
Q. What are the two mechanisms of injury?
Q. How do you classify fracture scaphoid?
Q. What are the clinical signs that help in diagnosing this fracture?
Q. What are the radiological views taken to diagnose fracture scaphoid?
Q. What are the methods of treating fracture scaphoid?
Q. Why is immobilization in 15° of dorsiflexion and 5° radial deviation preferred?
Q. What are the complications of fracture scaphoid?
Q. What is the cause for avascular necrosis?
Q. How do you treat complications of fracture scaphoid?
Q. Why are these fractures often missed?
Q. What is the course of action when fracture scaphoid is suspected clinically but there are no signs radiologically?

Essay Questions

Q. Describe the mechanism of injury of fracture scaphoid. Discuss the diagnosis and management of fracture scaphoid. Enumerate its complications.
Q. Discuss the complications of fracture scaphoid, their diagnosis and management.

Fall on an Outstretched Hand

Fall on an outstretched hand has a definite mechanism of injury. Many injuries can occur with a fall on an outstretched hand. They depend upon the magnitude and direction of the force, the age and quality of the bone as well as the position of the wrist, elbow and shoulder at the time of fall.

They are listed in order as follows from distal to proximal

1. Scaphoid fracture
2. Colles' fracture
3. Fracture of the radius and ulna
4. Posterior dislocation of the elbow joint
5. Supracondylar fracture
6. Fracture shaft of the humerus
7. Fracture neck of the humerus
8. Fracture of the clavicle

If there are associated forces such as valgus/varus stress, tortional stress, hyperpronation, hyperextension, etc. along with the fall on an outstretched hand, other injuries occur.

Colles' Fracture

Definition (Based on Original Description by Sir Abraham Colles)

It is a fracture occurring approximately within an inch and half of the inferior articular surface of the radius, with or without fracture of the ulnar styloid process, with or without subluxation/dislocation of the inferior radioulnar joint (Figs 2.13 and 2.14).

Who Described Colles' Fracture?

It was described by Sir Abraham Colles in the year 1814. Hence, it is known as Colles' fracture.

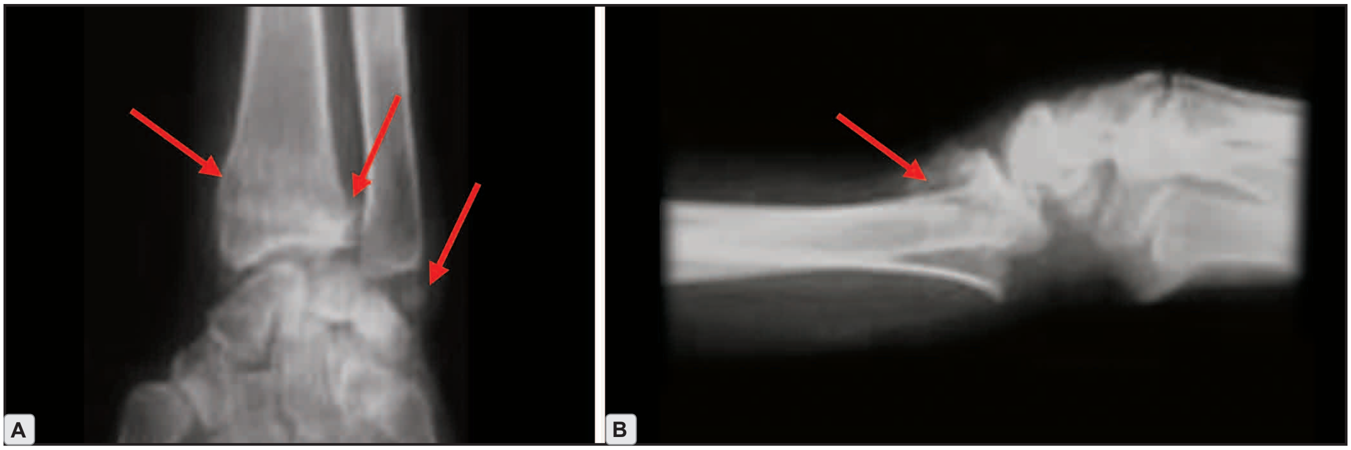
Mechanism of Injury

Mechanism of injury is by means of a fall on an outstretched hand (Fig. 2.15). The brunt of the force is on the thenar eminence of the palm and is transmitted to the lower radius. This causes the fracture.

Common Age Group

This injury is commonly seen in elderly, especially in women with osteoporosis.

Note: *In children, a fall on an outstretched hand causes an epiphyseal injury and not a Colles' fracture.*



Figures 2.13A and B

(A) PA (posteroanterior) and (B) Lateral view showing Colles' fracture. Note the classical site, displacements, dislocation of inferior radioulnar joint, fracture of the ulnar styloid process and the soft tissue shadow showing the typical 'Dinner fork deformity'.

Distal radius fracture-based on Frykman classification

Figure 2.14

Different types of distal radius fracture based on Frykman classification. Type I and II are extra-articular fractures. Type III and IV are fractures that involve radiocarpal joint. Type V and VI are fractures that involve radioulnar joint. Type VII and VIII are those involving both radiocarpal and radioulnar joint. Type I, III, V and VII are not associated with ulnar styloid process fracture. Type II, IV, VI and VIII are associated with ulnar styloid process fracture.

Figure 2.15

Colles' fracture is usually due to a slip and fall on the outstretched hands in elderly females.

Classical Deformity

The classical deformity is known as 'Dinner Fork' deformity (Figs 2.16A and B).

Displacements Seen in Colles' Fracture

The classical displacements seen in Colles' fracture are dorsal, lateral and proximal. In addition, the displaced fragment can rotate, angulate or tilt either dorsally (dorsal rotation/angulation/tilt) or laterally (lateral rotation/angulation/tilt). Impaction and supination are the other displacements seen in a few cases (Figs 2.17A and B).

Note: *Supination is an extreme form of displacement which is seen in compound Colles' fracture, when proximal radius projects out through the wound and distal radius rotates into supination.*

Treatment

A. *Nonoperative:* Closed manipulation and reduction under general anesthesia followed by immobilization in a plaster of Paris or fiber below elbow cast, moulded in 5° flexion and 5° ulnar deviation (Figs 2.18A and B). Duration of immobilization is 4–6 weeks.

A

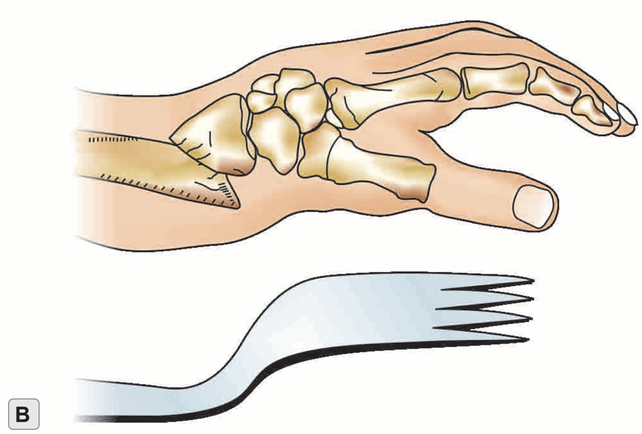
B

Figures 2.16A and B

AP and lateral views showing malunited Colles' fracture. Note the subluxation of the inferior radioulnar joint as seen by the proximal migration of the radial styloid process and the classical 'Dinner fork deformity'. *Normally the radial styloid process is at a lower level than the ulnar styloid process by about 1/2 an inch or 1.25 cms. In a Colles' fracture both are at the same level or the radial styloid is at a higher level than the ulnar styloid process. This happens because of proximal displacement of the fractured lower radius.*

Why is below elbow cast preferred and not above elbow cast?

1. Fracture remains stable when the cast is moulded in 5° of flexion and 5° of ulnar deviation. This eliminates the need to immobilize the joint above, i.e. elbow joint.
2. It prevents stiffness of the elbow as a consequence of immobilization.



Figures 2.17A and B
Classical 'Dinner fork' deformity of a Colles' fracture, clinical picture and diagrammatic representation.

Figures 2.18A and B
Position of moulding of below elbow cast /Colles' cast in 5° flexion and 5° ulnar deviation.

Indication for above elbow cast
Above elbow cast is indicated when comminution is observed and loss of reduction is suspected during the course of treatment.

B. *Operative:* Surgery is done in the form of closed Kirschner wire fixation under 'C' arm image intensifier control when there is dorsal comminution and articular surface involvement.

Note: *When Sir Abraham Colles described the fracture in the year 1814 the injury was generally of a low velocity in nature. Therefore, comminuted fractures were unlikely to occur. Further, there was no X-ray facility available to know the exact nature of the injury. Hence, these days it is recommended that comminuted articular fractures of the lower radius are not to be considered as Colles' fractures according to the definition. Strictly speaking, these are 'Intra-articular fractures of the lower radius'. Mechanism of injury is somewhat different in these fractures. A separate classification system exists for fractures of the lower radius (for clarification refer 'Fernandez and Jupiter' mechanistic classification).*

Complications

1. Malunion
2. Delayed union
3. Nonunion (rare)
4. Sudeck's atrophy
5. Shoulder hand syndrome
6. Carpal tunnel compression of median nerve
7. Rupture of extensor pollicis longus tendon

Treatment of Complications

Malunion: In malunion there is a deformity. It is treated by:

1. *Corrective osteotomy:* By using an osteotome the bone is cut at the site of deformity, *the deformity is corrected* and stabilized internally by means of an implant (plates and screws or K-wire) or externally by means of a plaster cast. Thus cosmesis is achieved by correcting the deformity.
2. *Darrach's procedure (for improving movement):* 6 cm skin incision is made over the lower third ulna, 4 cm incision in the periosteum and 2 cms of lower ulna is excised (6, 4, 2). *The procedure does not correct the deformity but masks the deformity.* A false joint develops which allows full range of pronation and supination. The procedure also relieves pain.

Delayed union and nonunion: Managed by means of 'Bone grafting' with or without internal fixation.

Sudeck's atrophy and shoulder hand syndrome: Managed by physiotherapy and adequate drugs for 'Sympatholysis'. Prevention of this complication is always better (refer Chapter 1).

Carpal tunnel compression of median nerve: Carpal tunnel decompression is the treatment advocated.

Extensor pollicis longus tendon rupture: Reconstruction by means of tendon transfer using extensor indicis as the donor tendon.

Revision Questions

Q. Define Colles' fracture.
Q. Why is it called "Colles' fracture"?
Q. What is the mechanism of injury?
Q. In which age group is this injury commonly seen?
Q. What is the injury occurring in the young by a similar fall on an outstretched hand?
Q. What is the classical deformity seen in Colles' fracture?
Q. What are the classical displacements seen in Colles' fracture ?
Q. When do you see supination deformity?
Q. How do you treat Colles' fracture?
Q. Why is below elbow cast preferred and not above elbow cast?
Q. When do you do surgery in Colles' fracture?
Q. When an above elbow cast is preferred in Colles' fracture?
Q. What are the complications of Colles' fracture?
Q. How will you manage complications of Colles' fracture?

Essay Questions

Q. Define Colles' fracture. Discuss the mechanism of injury, signs and symptoms, diagnosis and management of Colles' fracture.
Q. What are the complications of Colles' fracture? Discuss their management.

Smith's Fracture

Definition

Smith's fracture is defined as a transverse fracture of the lower radius within an inch of the articular surface with volar displacement of the fractured fragment (Also known as reverse 'Colles' fracture') (Figs 2.19A to C).

Figures 2.19A to C

(A and B) Classical volar displacement of the lower radius carrying along with it the carpus, metacarpals and phalanges. This gives rise to a classical 'Garden spade' deformity. Also see (C) the clinical photograph of the deformity.

Who Described Smith's Fracture?

It was described by Robert William Smith (1807–1873), an Irish surgeon in the year 1841.

Mechanism of Injury

The mechanism of injury is by means of a fall on a flexed wrist.

Classical Deformity

It is the "Garden Spade" deformity (reverse of "Dinner Fork" deformity).

Treatment

A. *Operative:* This is an unstable fracture. Hence, it is treated by open reduction and internal fixation using a specific type of plate known as "Buttress Plate" (Ellis Buttress Plate).
B. *Nonoperative:* Employed only in minimally displaced fractures. Following manipulation and reduction, an above elbow cast is given for immobilization which is continued for a period of 6 weeks with a periodic check for loss of reduction. If loss of reduction is observed, an open reduction and internal fixation (ORIF) is done at the earliest, within 2 weeks.

Complications

1. Malunion
2. Subluxation of inferior radioulnar joint
3. Sudeck's atrophy

Treatment of Complications

The complications are treated in a similar manner as described in Colles' fracture.

Revision Questions

Q. What is the definition of Smith's fracture?
Q. Who described "Smith's fracture"?
Q. What is the mechanism of injury?
Q. What is the classical deformity seen?
Q. How do you treat "Smith's fracture"?
Q. Is there a role for closed manipulation, reduction and a plaster of Paris or fiber cast immobilization?
Q. What are the common complications?

Essay Question

Q. Define Smith's fracture. Discuss the mechanism of injury, clinical signs, diagnosis and management of Smith's fracture.

Barton's Fracture

Definition

Barton's fracture is defined as an articular marginal fracture involving the dorsal 1/3rd or volar 1/3rd articular surface of the lower radius with subluxation or dislocation of the carpus.

Who Described "Barton's Fracture"?

It was described by John Rhea Barton, an American surgeon in the year 1814.
Note: *Original article describes only dorsal Barton's fracture. Volar Barton's fracture was recognized later.*

Mechanism of Injury

Dorsal Barton's fracture: The mechanism of injury is an extreme dorsiflexion occurring at the wrist accompanied by a pronating force resulting in shear and a dorsal articular marginal fracture (Figs 2.20A and B).

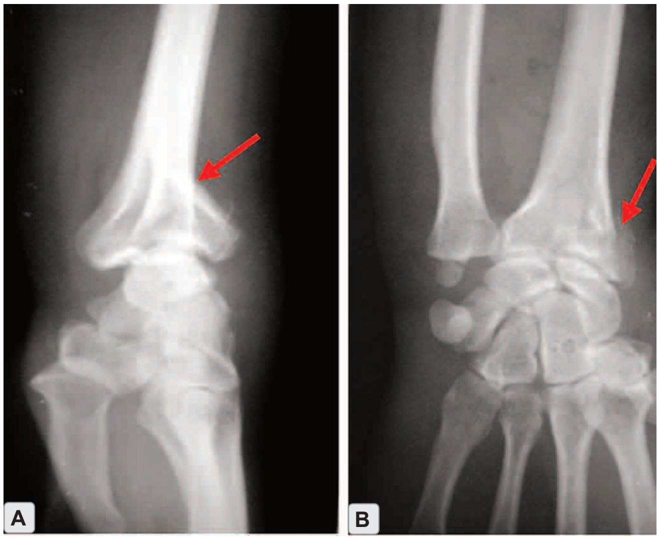
Volar Barton's fracture: The mechanism of injury is an extreme palmar flexion occurring at the wrist accompanied by a supinating force resulting in shear and a volar articular marginal fracture (Figs 2.21A to D).
Note: *Clinical diagnosis is difficult. It can be easily mistaken for Colles' fracture (Dorsal Barton's) or Smith's fracture (Volar Barton's). X-ray is confirmatory.*

Treatment

A. *Operative:* This fracture is treated by open reduction and internal fixation using special plate known as Buttress plate (e.g. Ellis Buttress plate, Jupiter plate).
B. *Nonoperative:* Has no role except in very selected cases of incomplete fracture. Complete fractures invariably get displaced sooner or later because of the pull of the carpal ligament and are treated surgically.

Complications

Following are the complications seen:
1. Malunion with angulation and radial shortening
2. Distal radioulnar joint (DRUJ) subluxation
3. Sudeck's atrophy
4. Rupture of extensor pollicis longus tendon (Dorsal Barton's)
5. Radiocarpal degenerative arthritis (late complication)



Figures 2.20A and B

PA and lateral views showing a dorsal Barton's fracture.

Figures 2.21A to D

Volar Barton's fracture pre- and postoperative. Note classical displacements. A T-buttress plate has been used to fix the fracture.

Revision Questions

- Q. Can Barton's fracture be clinically diagnosed?
- Q. What is the mechanism of injury for a volar Barton's fracture?
- Q. What is the mechanism of injury for a dorsal Barton's fracture?
- Q. Why it is called "Barton's fracture"?
- Q. Define Barton's fracture.
- Q. How will you treat Barton's fracture?
- Q. Is there any role for nonoperative method with casts?

Essay Question

- Q. Define Barton's fracture. Discuss the diagnosis and management of Barton's fracture. Enumerate its complications.

Essex-Lopresti Fracture

Definition

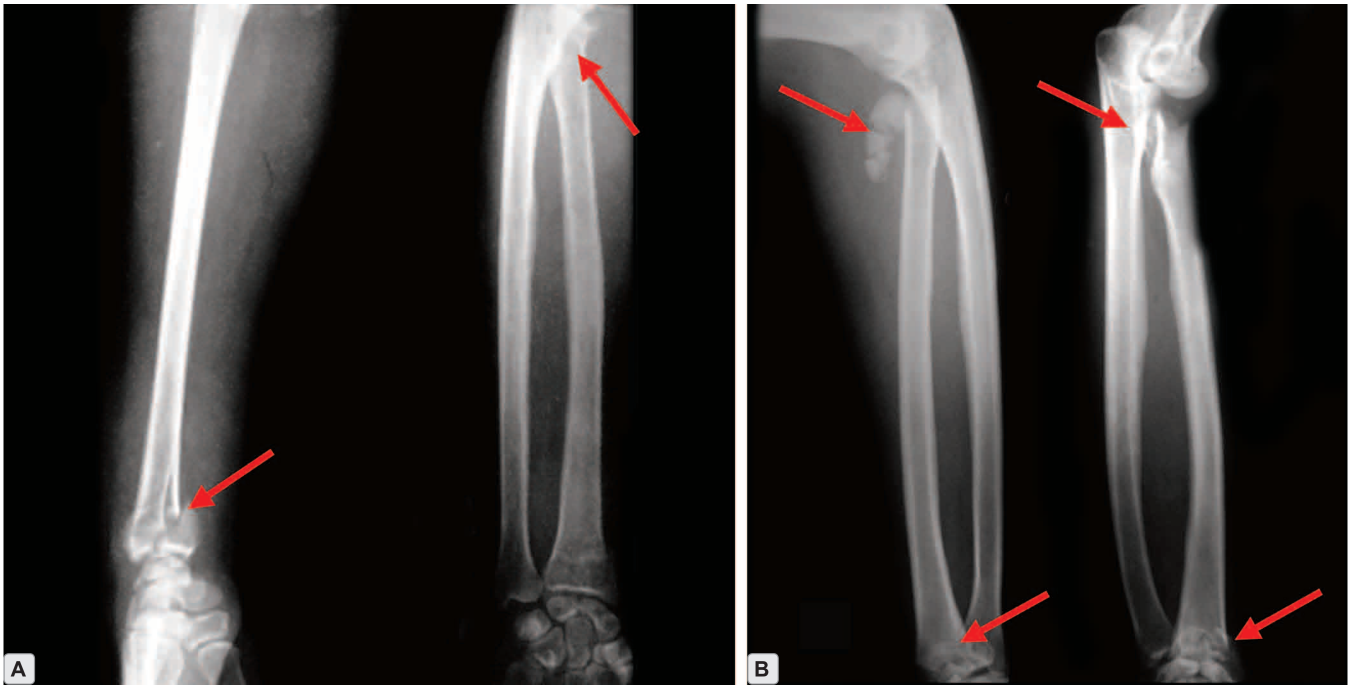
It is a comminuted fracture of the radial head with dislocation/subluxation of the distal radioulnar joint (DRUJ). Described by Essex-Lopresti in 1951 (Figs 2.22A and B).

Mechanism of Injury

Mechanism of injury is by means of a fall from a height on an acutely dorsiflexed wrist resulting in axial loading of the radius. This longitudinal compression force, disrupts the interosseous membrane and causes the above injury.

Clinical Importance

1. This injury should be suspected in every case of radial head fracture and the inferior radioulnar joint is examined both clinically and radiologically for subluxation.
2. The head of the radius in these injuries, is never excised immediately without stabilization of the inferior radioulnar joint as it will aggravate the subluxation of the inferior radioulnar joint.



Figures 2.22A and B

Essex-Lopresti injury, as a result of severe form of axial loading resulting in inferior radioulnar subluxation, comminuted fracture of the head of the radius as well as comminuted articular fracture of the lower radius.

Treatment

1. Inferior radioulnar joint reduction is achieved primarily by supinating the forearm and if needed stabilized with a K-wire. Interosseous membrane takes about 6 weeks for healing.
2. Fractured head of the radius is managed next by open reduction and internal fixation (ORIF) or excision depending on the nature of injury.

Essay Question

Q. Define Essex-Lopresti fracture. Discuss the mechanism of injury and management.

Chauffeur's Fracture

It is a fracture of the radial styloid process. It used to occur among Chauffeur's in olden days, when the crank which they used to start the car kicked back and hit the thenar side of the wrist.

Shearing force generated by the carpus is responsible for this fracture (Fig. 2.23).

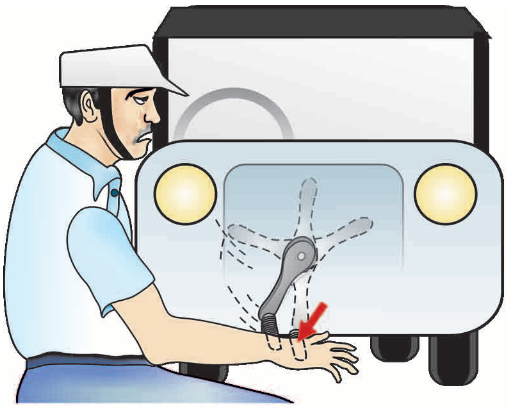


Figure 2.23

Mechanism of injury of a Chauffeur's fracture.

Treatment

This fracture is usually unstable because of the pull of the brachioradialis and strong radiocarpal ligament. Hence, internally fixed using 'Kirschner wire' (K-wire).

FRACTURES IN THE FOREARM

General Information

In forearm, fractures of both the bones, that is fractures of radius and ulna, occur more commonly than a single bone fracture.

A fracture of the single bone fracture of the forearm, rarely occurs without subluxation or dislocation of a joint above or a joint below. So, one must examine the superior and inferior radioulnar joints in a patient with a forearm fracture and take X-rays including elbow and the wrist based on the findings.

The common fractures occurring in the forearm are:

1. Fracture of the radius and the ulna
2. Monteggia fracture
3. Galeazzi fracture

Fracture of the Radius and the Ulna

Mechanisms of Injury

It occurs by means of a:

1. Direct blow onto the forearm.
2. Fall on an outstretched hand with forearm pronated.

Deforming Forces (Figs 2.24A to C)

a. Proximal 1/3rd fractures (Above the insertion of pronator teres). The proximal fragment is flexed and supinated because of the unopposed action of biceps brachii and supinator and distal fragment is pronated because of the action of pronator teres and pronator quadratus.
b. Middle 1/3rd fractures and lower 1/3rd fractures (below the insertion of pronator teres). The proximal fragment is in midprone position because the action of supinator and pronator teres balance and neutralize each other. The distal fragment is in pronation because of the action of pronator quadratus.

An understanding of the deforming forces is important in planning the treatment of these fractures. It dictates the alignment of distal fragment in relation to proximal fragment. In proximal 1/3rd fractures the distal fragment is aligned in supination. In middle 1/3rd fracture and lower 1/3rd fractures, the distal fragment is aligned in midprone position.

Treatment

These fractures are highly unstable. Except in children and adults with undisplaced fractures (with a check on redisplacement during closed treatment) there is no role for closed reduction and plaster cast immobilization. Open reduction and internal fixation is the treatment of choice. Plates and screws or intramedullary nails in selected cases, are implants that are used. *Special attention must be paid to maintain the radial bow and the interosseous space so as to get back the movements of pronation and supination (Fig. 2.25).*

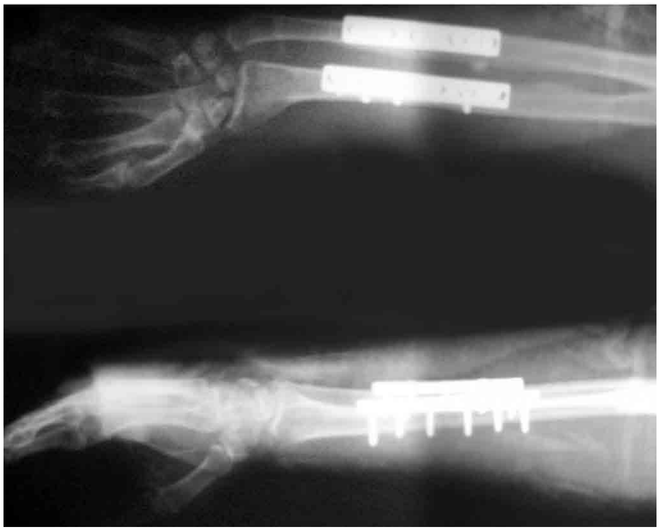


Figure 2.25

Internal fixation of fractures of both the bones of the forearm using DCP. Note the presence of old malunited Colles' fracture with subluxation of the distal radioulnar joint. These days DCP is not used. LC-DCP is the plate of choice.

Figures 2.24A to C

Classical deformity at different levels in forearm fractures; (A) Upper 1/3rd fracture; (B) Middle 1/3rd fracture; (C) Lower 1/3rd fracture.

Complications

1. Malunion
2. Nonunion
3. Cross union (synostosis) because of single hematoma formation

Revision Questions

Q. What is the mechanism of injury for fracture of both bones of the forearm?

Q. What are the deforming forces responsible for displacement and what is their importance?

Q. What is the treatment of this fracture?

Q. What are the complications of the fracture of both the bones of the forearm?

Monteggia Fracture

Monteggia fracture was described first by an Italian surgeon Giovanni Batista Monteggia, in Milan, in the year 1814, i.e. in the pre-Roentgen era without the help of X-rays.

Definition

Monteggia fracture is defined as the fracture of the proximal ulna with subluxation or dislocation of the superior radioulnar joint. When superior radioulnar joint gets dislocated, the radiohumeral component of the elbow joint, i.e. the radio-capitular articulation also gets dislocated.

Note: *Because of the above reason Monteggia fracture is considered to be more unstable than Galleazzi fracture.*

Radiological diagnosis of superior radioulnar joint dislocation/subluxation: It is diagnosed by the help of a line drawn along the long axis of the radius which is continued through the elbow joint. Normally, this line cuts the lateral condyle of the humerus in both AP and LAT views (Figs 2.26A to D). If it does not, it indicates subluxation or dislocation of the superior radioulnar joint.

Mechanisms of Injury (Fig. 2.27)

Following are the commonly associated mechanisms.

1. Fall on an outstretched hand with forced pronation.
2. Direct forceful blow to the forearm.

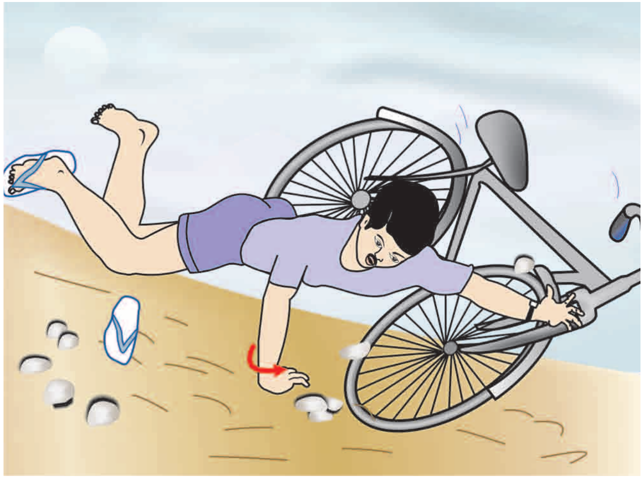
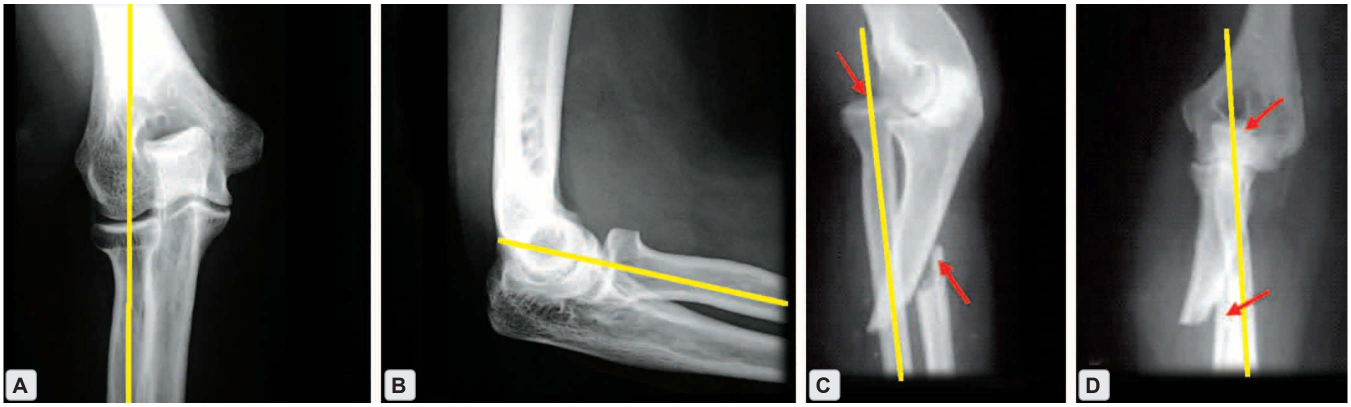


Figure 2.27

Diagrammatic representation of a hyperpronation injury which is responsible for 'Monteggia fracture'.



Figures 2.26A to D

(A and B) The maintained relation of superior radioulnar and radio-capitular joints as proven by a line drawn along the long axis of the radius which is continued through the elbow; (B and C) The relation is lost when there is superior radioulnar joint subluxation or dislocation ('Monteggia fracture').

Classification

It is classified using 'Bado's classification' as follows (Figs 2.28A to D).

Type I Fracture of the upper or middle third of the ulna with anterior displacement of the head of the radius.

Type II Fracture of the upper or middle third of the ulna with posterior displacement of the head of the radius.

Type III Fracture of the upper or middle third of the ulna with lateral displacement of the head of the radius.

Type IV Fracture of upper or middle third of ulna and radius with anterior displacement of the head of the radius.

Hume's Fracture

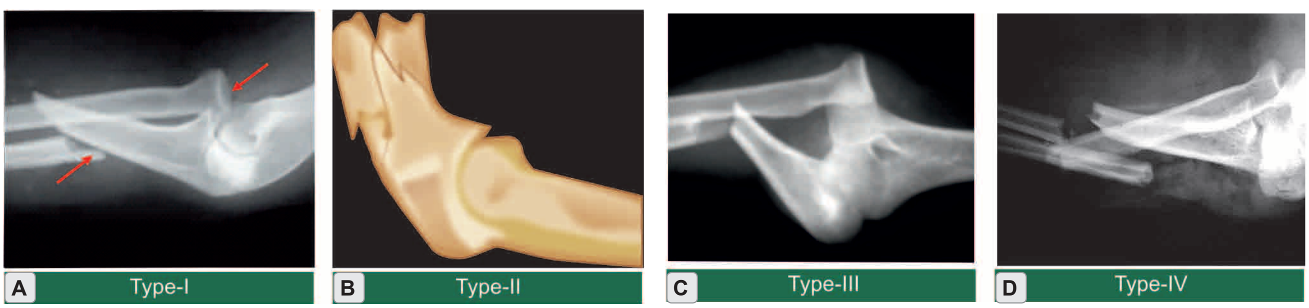
It is a *high Monteggia fracture*, commonly occurring in children and one of the Monteggia variants (Figs 2.29A to D).

Treatment

Operative: The fracture of the ulna, is always treated by open reduction and external fixation (OREF) in a stable manner. Due attention also given to the stable anatomical reduction of superior radioulnar joint. If needed a ligament repair/reconstruction is done primarily.

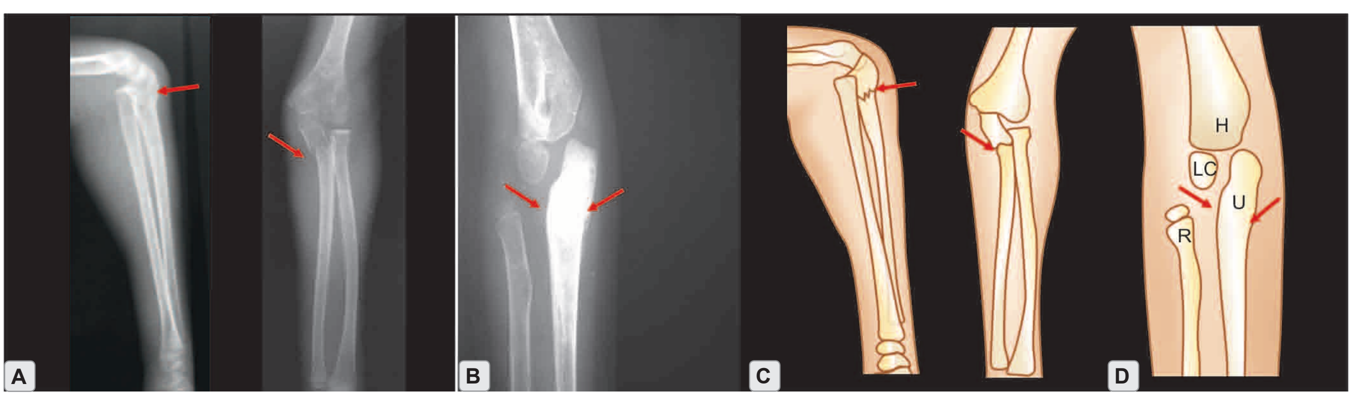
Complications

1. Malunion (Fig. 2.30)
2. Nonunion
3. Redislocation
4. Radioulnar synostosis
5. Chronic pain
6. Nerve injuries



Figures 2.28A to D

Monteggia fracture—based on Bado's classification.



Figures 2.29A to D

(A and B) Pediatric Hume's fracture. (C and D) Diagrammatic representation of the same injury. Figure A is a recent injury. Figure B old negelected injury with malunion along with persistent superior radioulnar joint dislocation. The latter needs osteotomy of the malunited ulna followed by reduction and stabilization of the superior radioulnar joint dislocation along with internal fixation of the osteotomy.
(Humerus—H; Radius—R; Ulna—U; Lateral condyle—LC)

Figure 2.30

An old unreduced Hume's fracture dislocation. Note the persistent dislocation of the superior radioulnar joint, atrophy of the head of the radius and the malunion at the upper 1/4th of the ulna. The patient has adapted very well to the changed structure of the elbow and has no complaints except cosmesis.

Revision Questions

Q. Who described Monteggia fracture?

Q. What is Monteggia fracture?

Q. In a Monteggia fracture, which other joint gets dislocated along with superior radioulnar joint dislocation ?

Q. How will you diagnose superior radioulnar joint subluxation or dislocation radiologically?

Q. What is the mechanism of injury in a Monteggia fracture?

Q. How do you classify Monteggia fracture?

Q. What is "Hume's fracture" ?

Q. What is the treatment of Monteggia fracture ?

Q. What are the complications of Monteggia fracture?

Essay Question

Q. Define Monteggia fracture. Discuss the mechanisms of injury, diagnosis and treatment of Monteggia fracture. Enumerate the complications.

Galeazzi Fracture

The Galeazzi injury pattern was first described by Sir Astley Cooper in the year 1842, exactly 92 years before Ricardo Galeazzi, an Italian Surgeon at the Instituto de Rachitici Milan described the results of treatment in 18 cases in the year 1934. In 1941, William Campbell called this *as a fracture of necessity* because surgery is necessary for the treatment of this fracture and also named it as Galeazzi fracture. This fracture is also known as reverse Monteggia fracture (Fig. 2.31).

Definition

It is a fracture of the lower third or lower fourth of radius with subluxation or dislocation of distal radioulnar joint (DRUJ) (Figs 2.32A and B).

Note: *This fracture can occur in the radial shaft, anywhere between the bicipital tuberosity and approximately 5 cms from the distal articular surface of the radius. More proximal the fracture, lesser is the DRUJ instability. More distal the fracture, greater is the DRUJ instability.*

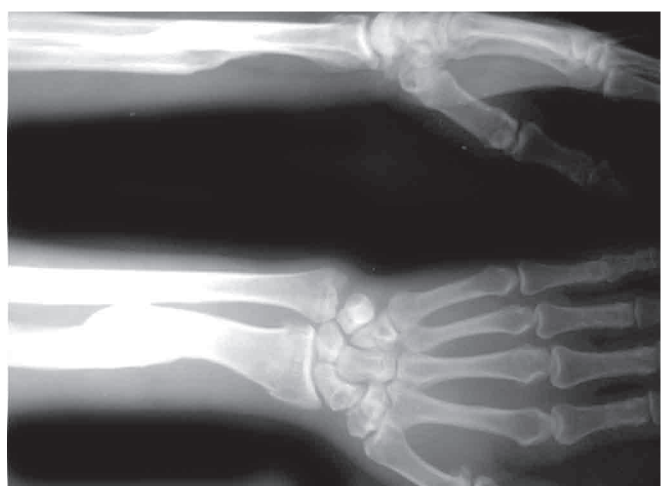
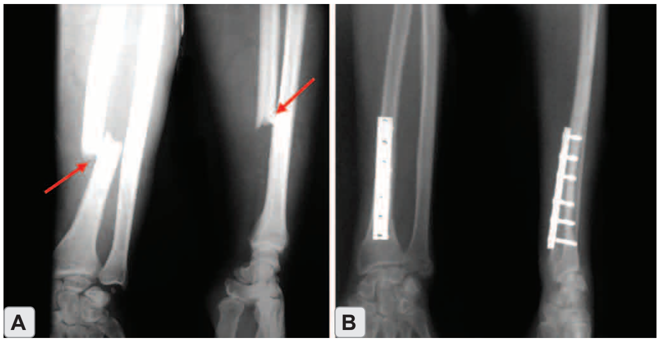


Figure 2.31

Malunited Galeazzi fracture with distal radioulnar joint (DRUJ) subluxation with radial deviation of the carpus as a consequence of non-operative treatment. For the very same reason, William Campbell called it as a fracture of necessity and advocated surgery.



Figures 2.32A and B

Galeazzi fracture pre- and postoperatively: (A) Galeazzi fracture; (B) ORIF with DCP. Note the importance of anatomic reduction of the fracture which automatically reduces the inferior radioulnar joint subluxation. Currently, LC DCP/LCP is the implant used.

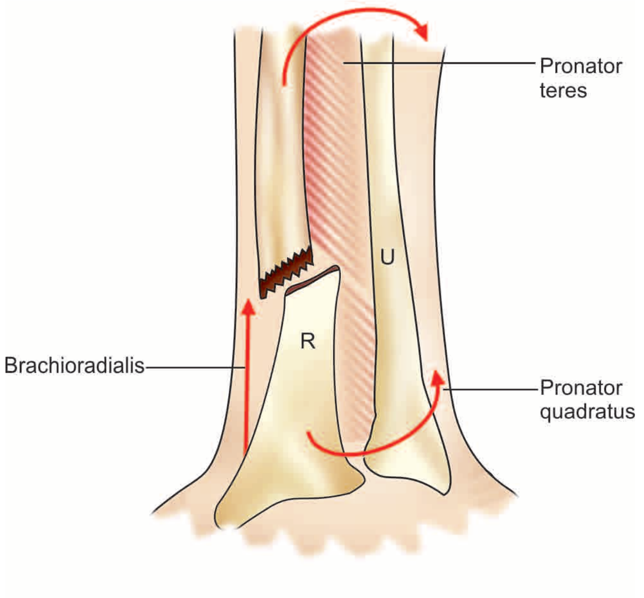


Figure 2.33

Muscular forces responsible for the displacement in a Galeazzi fracture (Radius—R; Ulna—U).

Mechanism of Injury

The mechanism of injury is an axial loading of radius in a hyperpronated forearm (Fig. 2.33).

Treatment

In adults: Open reduction and internal fixation using plates and screws is a necessity. For example, limited-contact dynamic compression plate (LC DCP). Distal radioulnar joint (DRUJ) has to be reduced always during the procedure of open reduction and internal fixation by closed methods and rarely by open methods.

In children: Closed manipulation, reduction and immobilization in plaster of Paris (POP) cast is the treatment.

Unlike adults, because the periosteum is thick and bones are elastic, the reduction is maintained.

Complications

1. Malunion
2. Nonunion
3. DRUJ instability

Revision Questions

Q. What is Galeazzi fracture?
Q. What is the mechanism of injury in a Galeazzi fracture?
Q. What is the treatment of a Galeazzi fracture?
Q. What are the complications?

Essay Question

Q. Define Galeazzi fracture. Discuss the mechanism of injury, diagnosis and treatment of Galeazzi fracture. Enumerate the complications.

FRACTURES AROUND THE ELBOW

General Information

Elbow is a hinge joint with humeroradial and humeroulnar articulations. Before skeletal maturity, trauma causes epiphyseal injuries and after skeletal maturity, fractures of the ossified components. The names of the fractures are derived accordingly.

Among the fractures of the medial and the lateral epicondyles, medial epicondyle is commonly fractured because its epiphysis remains as a separate epiphysis till skeletal maturity and then fuses with the shaft of the humerus. Whereas, the remaining epiphyses of the lower end of the humerus, namely, the trochlea, the capitulum and the lateral epicondyle, fuse to form a single epiphysis (Lateral Condyle) at puberty which collectively fuses with the shaft of the humerus at skeletal maturity. For the same reason, lateral condylar fractures are common and not the fractures of the medial condyle. A fracture which occurs above the condyles is known as supracondylar fracture. A fracture which splits the condyles into two is known as intercondylar fracture.

The common fractures around the elbow joint are:
1. Radial head fracture
2. Olecranon fracture
3. Capitulum and trochlear fracture
4. Medial epicondyle fracture
5. Lateral epicondyle fracture
6. Medial condyle fracture
7. Lateral condyle fracture
8. Supracondylar fracture
9. Intercondylar fracture

Radial Head Fracture

General Information

Head of the radius forms a major component of superior radioulnar as well as radiohumeral articulation. Hence proper management of fractured radial head is very essential in order to preserve the joint function. Many a times it may be associated with elbow dislocation and before examining, the elbow dislocation would have reduced by itself. Hence, it is missed easily. A simple fracture of the head of the radius will not cause excessive swelling. Hence, when excessive swelling is present, one should always suspect a possibility of a reduced elbow dislocation along with radial head fracture.

Classification (Figs 2.34 to 2.36)

Based on Mason's classification, it is classified as:

Type I	Undisplaced.
Type II	Displaced, single fragment fracture less than 1/3rd of the articular surface.
Type III	Displaced, involving more than 1/3rd of the articular surface or a comminuted fracture.

Mechanism of Injury

Is by a fall on an outstretched arm with elbow in extension. The compression stress is transmitted along the axis of the radius and the radial head hits the capitulum of the humerus thereby causing the fracture.

Treatment

Radial head facture may be part of elbow dislocation. This fact should always be kept in mind during management of radial head fractures.

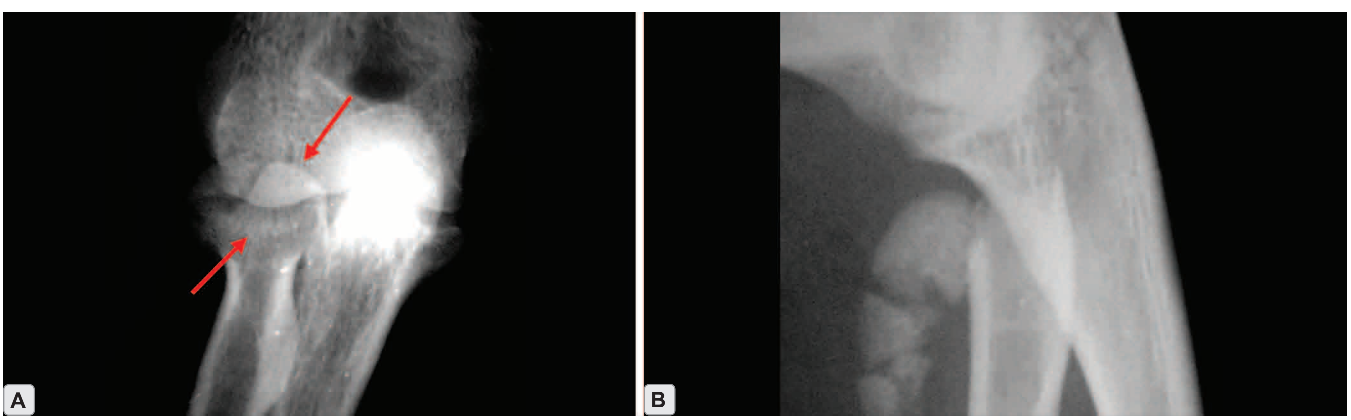
Undisplaced and incomplete fractures are managed by immobilization in a plaster cast or a slab for a period of three weeks followed by removal and mobilization.

Comminuted, displaced fractures are managed by excision of the head of the radius. Displaced noncomminuted fractures are managed by open reduction and internal fixation or by excision of the head of the radius, depending on the size of the fragment.

Rule of Three: Less than 1/3rd involvement, less than 30° angulation, less than 3 mm displacement of the radial head, qualifies for non-operative management. If any of these three criteriae are not met, surgery is the treatment of choice.

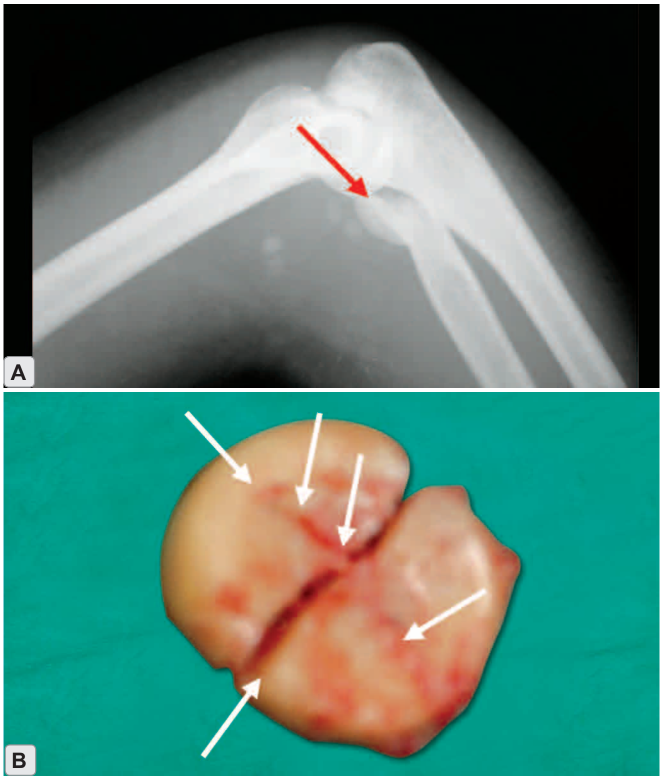
Complications

1. Avascular necrosis of the head of the radius.
2. Malunion with restriction of pronation and supination.
3. Painful limitation of pronation and supination.



Figures 2.34A and B

(A) Type II and (B) Type III fracture of the head of the radius based on Mason's classification.



Figures 2.35A and B

A three weeks old fracture Mason's type III involving the head of the radius. Patient underwent excision of the head of the radius. Note the two major fragments with split in the fragments. Also note the edges of the fracture which have become rounded.

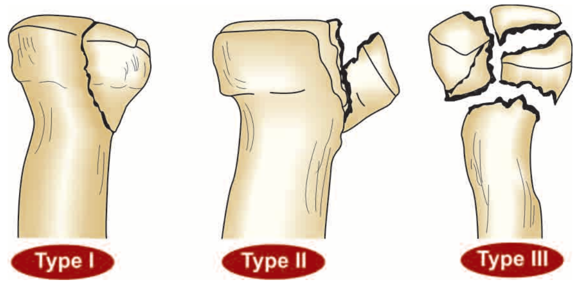


Figure 2.36

Classification of radial head fractures as classified by Mason (refer text).

Revision Questions

Q. How do you classify radial head fracture?

Q. What is the mechanism of injury in fracture head of the radius?

Q. How is this fracture managed?

Q. Name the complications of radial head fracture.

Olecranon Fracture

General Information

Olecranon fractures are seen in a diverse group of injuries ranging from a simple undisplaced fracture to a complex fracture dislocation of elbow joint.

Mechanisms of Injury

Three mechanisms have been identified.

1. Avulsion fracture. This occurs when a person falls on a semiflexed elbow with forearm in supination. Under such circumstance, the triceps contracts against the resistance of a flexed elbow. This results in avulsion fracture of the olecranon (Fig 2.37)
2. Direct impact onto the olecranon by a fall on the point of the elbow or by a direct blow.
3. A hyperextension injury.

Classification

Based on Schatzker classification:

Type A	Simple transverse fracture
Type B	Transverse impacted fracture
Type C	Oblique fracture
Type D	Comminuted fracture
Type E	More distal fracture, extra-articular
Type F	Fracture dislocation

Figure 2.37

An oblique fracture of the olecranon process of the ulna.

Based on Mayo clasification:

Type I: Non-displaced fractures
- Type I A—Noncomminuted
- Type I B—Comminuted

Type II: Displaced, stable fractures

The proximal fracture fragment is displaced more than 3 mm, but the collateral ligaments are intact and there is no elbow instability
- Type II A—Noncomminuted
- Type II B—Comminuted

Type III: Displaced, unstable fractures

The proximal fracture fragment is displaced and the forearm is unstable in relation to the humerus. These are fracture-dislocations
- Type III A—Noncomminuted
- Type III B—Comminuted

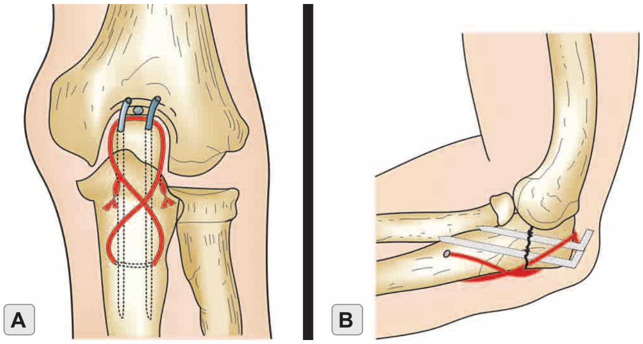
Treatment

Nonoperative:

This treatment is meant for Mayo Type I fractures with intact extensor mechanism. These fractures are immobilized only for a short period of 7–10 days for relief of pain. Afterward, the elbow is gradually mobilized. As the extensor mechanism is intact, mobilization does not cause displacement.

Operative:

Noncomminuted, transverse and stable fractures (Figs 2.38A and B):
- Treatment of choice is open reduction and internal fixation using the technique of tension band wiring, which converts the distraction force of triceps pull into a compression force and promotes healing.
- Alternatively, a single long AO cancellous bone screw can also be used as an intramedullary device for fixation.



Figures 2.38A and B

'Tension band' wiring of fracture olecranon.

Comminuted and unstable fractures:

Fixation is done using plates and screws, e.g. Hookplate Recon plate, Locking Recon plate, etc.

Revision Questions

Q. What are the mechanisms of injury in olecranon fracture?

Q. Classify olecranon fractures.

Q. What is the treatment of olecranon fracture?

Capitulum and Trochlea Fractures

These are rare fractures involving the articular surface of the lower humerus. They need open reduction and internal fixation to restore articular surface congruity. Mini-screws, 'Herbert screws' and sometimes 'Kirschner wires' are used to fix these fractures.

Medial and Lateral Epicondyle Fractures

These are avulsion fractures which occur due to the pull of common flexor and extensor muscle origin respectively. They have to be fixed firmly and immobilized in a cast or a slab till union occurs. In adults, sometimes the medial epicondylar fragment is excised and ulnar nerve is transposed anteriorly. In children medial epicondyle fracture is considered as a type of epiphyseal injury and is always fixed anatomically (Fig. 2.39).

Figure 2.39

Medial epicondyle fracture. It is an avulsion fracture occurring because of the pull of common flexor origin.

Medial and Lateral Condyle Fractures

General Information

Lateral half of trochlea, capitulum and lateral epicondyle constitute the *lateral condyle*.

Medial half of trochlea with medial epicondyle constitute the *medial condyle*.

Stimson in 1883 first described the lateral condyle fracture in his book *Treatise on fractures*. Milch in 1955 recognized the significance of these fracture patterns in elbow stability and classified them.

Mechanisms of Injury

a. Lateral condyle fractures
   Common mechanism: A severe varus stress results in fracture of the lateral condyle because of 'pull off' by common extensor origin.
   Alternate mechanism: Fall on an extended hand resulting in a compressive impact of the radial head onto the lateral condyle—'push off' theory.
b. Medial condyle fractures
   Common mechanism: A severe valgus stress results in fracture of the medial condyle because of 'pull off' by common flexor origin.
   Alternate mechanism: A fall on the point of the elbow resulting in a direct blow onto the posteromedial aspect of the medial condyle.

Classifications

Lateral condyle fracture: Based on Milch anatomical classification (1964)

Type I — Extends through the ossification center, exits at the radiocapitular groove with intact lateral crista of trochlea (Salter and Harris Type IV Epiphyseal Injury—Refer chapter on fractures in children). The elbow is relatively stable.

Type II — Extends across the physis, exits through the trochlea fracturing the lateral crista of trochlea (Commonly Salter and Harris Type II. Sometimes can be Type IV Epiphyseal Injury). Results in unstable elbow.

Based on Jakob's classification (1975) (Figs 2.40A to C)

Stage I — Nondisplaced with intact articular surface.

Stage II — Moderate rotational displacement with fractured articular surface.

Stage III — Complete displacement with rotation and elbow instability.

Medial Condyle Fracture

Milch anatomical classification (1964)

Type I — Splits the trochlear groove, lateral trochlear ridge intact.

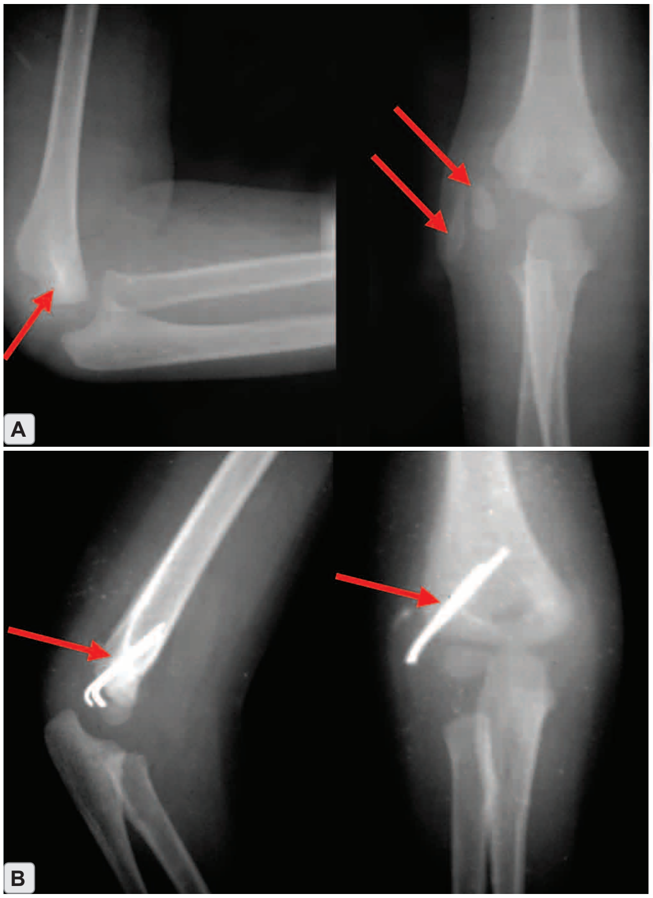
Type II — Spilts the capitotrochlear sulcus; trochlear ridge is part of the fracture.

Treatment

Since these injuries are commonly seen in children, involve the articular surface of the lower humerus and are epiphyseal injuries, they need open/closed reduction and internal fixation in order to restore articular surface congruity and prevent growth disturbance.

Figures 2.40A to C

Three stages of lateral condylar fracture according to Jakob's classification: (A) Stage-I; (B) Stage-II; (C) Stage-III.



Figures 2.41A and B

(A) Grossly displaced lateral condylar fracture. Note that the fractured surface is rotated and is facing outwards. Accurate reduction is necessary to restore function. (B) After open reduction and internal fixation with the help of two 'Kirschner wires'.

Operative: Both medial and lateral condyle fractures are treated by open reduction and internal fixation using K-wires. Plates and screws are used in older age group as per indication (Figs 2.41A and B).

Complications of Lateral Condyle Fracture

1. Nonunion
2. Malunion with deformity
3. Limitation of movement of the elbow
4. Tardy ulnar nerve palsy

   'Tardy ulnar nerve palsy' means, delayed ulnar nerve paralysis. Nonunion of the lateral condyle causes a progressive valgus deformity at the elbow. This stretches the ulnar nerve gradually, resulting in a progressive ulnar nerve palsy.

Treatment: Treatment for 'Tardy ulnar nerve palsy' is anterior transposition of the ulnar nerve. At the same time lateral condyle is fixed in the best possible manner.

Revision Questions

Q. What is a lateral/medial condyle?
Q. Who described lateral condyle fracture first?
Q. What are the mechanisms of injury for a lateral condyle fracture?
Q. What are the mechanisms of injury for a medial condyle fracture?
Q. In which age group these fractures are commonly seen?
Q. What is the treatment for lateral/medial condyle fracture?
Q. Name the complications of lateral condyle fracture.
Q. What is 'Tardy ulnar nerve palsy'?
Q. What is the treatment for 'Tardy ulnar nerve palsy'?

Supracondylar Fracture of the Humerus

This fracture occurs just above the two condyles of the lower humerus. Hence, derives the name supracondylar fracture.

Age

Commonly seen between 5-10 years.

Types

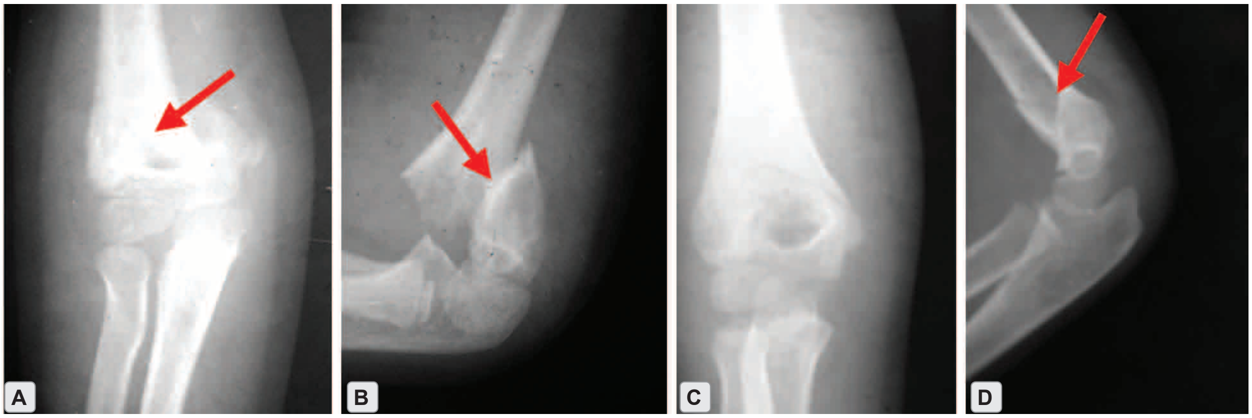
Two types are described (Figs 2.42A to D).

1. Extension type
2. Flexion type

It is the extension type that is commonly seen. Flexion type is not commonly seen.

Mechanism of Injury

a. *Extension type:* By means of a fall on an outstretched hand with the elbow in extension or minimal flexion. The force is transmitted through the supracondylar portion of the humerus (Fig. 2.43).
b. *Flexion type:* By means of a fall on the point of a fully flexed elbow.



Figures 2.42A to D

(A and B) Extension type of supracondylar fracture; (C and D) Flexion type of supracondylar fracture. *Note the site of the fracture and direction of the fracture line. The site of the fracture is just above the condyles. The directions of fracture lines are opposite to each other. In* ***extension*** *type it is* ***obliquely upwards and backwards****. In* ***flexion*** *type it is* ***obliquely upwards and forwards.***

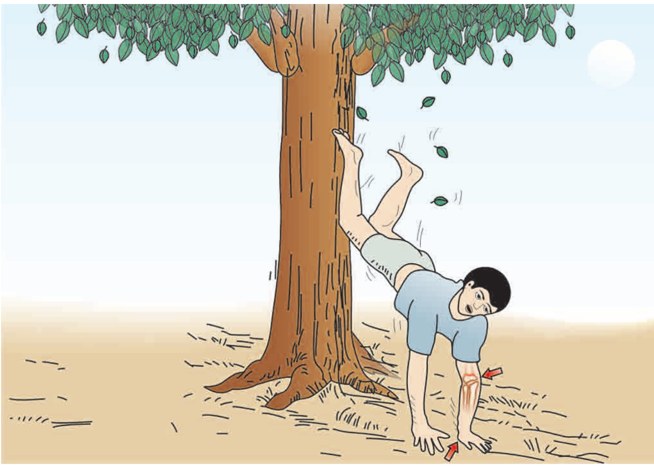


Figure 2.43

The mechanism of injury of an extension type of supracondylar fracture.

Displacements: Displacements seen are as follows:

- *In extension type:* Posterior, medial/lateral and rotation.
- *In flexion type:* Anterior, medial/lateral and rotation.

Nature of the fracture line and its importance

- In extension type, it is directed obliquely upwards and backwards.
- In flexion type, it is directed obliquely upwards and forwards.

The nature of the fracture line is of importance in the treatment of supracondylar fracture.

An extension type of supracondylar fracture is stable in flexion and hence after reduction, it is immobilized in flexion. It is unstable in extended position of the elbow because of the nature of the fracture line (Figs 2.42A and B).

A flexion type of supracondylar fracture is stable in extension and hence after reduction, it is immobilized in extension. It is unstable in flexed position of the elbow because of the nature of the fracture line (Figs 2.42C and D).

The role of triceps and brachialis muscle: These muscles play an important role in maintaining the reduction of this fracture.

The triceps muscle acts as an internal splint and helps in maintaining the reduction in extension type of supracondylar fracture when the elbow is immobilized in flexion. The brachialis muscle acts as an internal splint and helps in maintaining the reduction in flexion type of supracondylar fracture when the elbow is immobilized in extension.

Classification

Based on **Gartland** classification, supracondylar fractures are classified as follows:

Type I	Undisplaced
Type II	Displaced with some contact at the posterior cortex
Type III	Displaced with no cortical contact

Treatment

A. *Non operative methods:*

i. Closed manipulation and reduction with maintenance of reduction in a plaster slab. It is indicated mainly in Gartland's Type-I and Type-II fractures and of limited use in Type-III fractures. Generally Gartland's Type-III fractures are fixed with 'Kirschner's wires' after reduction.

ii. Skeletal traction: A K-wire is passed through the olecranon process and an overhead traction is applied (Fig. 2.44).

It is indicated under following circumstances.

a. Gross swelling at the elbow preventing manipulation
b. Poor skin condition with contusion and blistering
c. Ensuing vascular insufficiency along with the above.

B. *Operative:* Closed/open reduction and internal fixation using 'Kirschner Wires'. It is indicated in:

a. Badly displaced (Type-III), unstable supracondylar fracture
b. Open supracondylar fracture
c. Open or closed supracondylar fracture with vascular injury which needs vascular repair (Fig. 2.45).

It is also done selectively in Gartland Type-II when early mobilization is indicated.

In closed fractures, this fixation can be done by a closed method using image intensifier or by minimally opening the fracture site, medially and laterally. It is not advisable to split the triceps muscle for exposing the fracture. Splitting of the triceps muscle, always results in residual stiffness and prolongs the period of mobilization. Hence, should be done only when it is absolutely necessary.

Baumann's Angle (Fig. 2.46)

In children, it is difficult to assess the accuracy of reduction of varus. The Baumann's angle helps to determine the accuracy. It is an angle formed between a line along the long axis of the humerus and a line along the coronal axis of the capitular physis. Normal angle is around 80°. If the angle increases, it indicates the varus position of the distal fracture fragment.

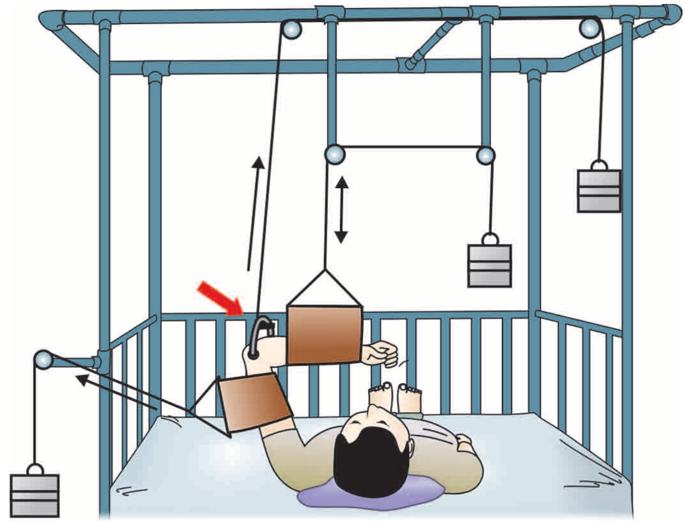


Figure 2.44

Treatment of supracondylar fracture by traction with help of a Balkan beam frame.

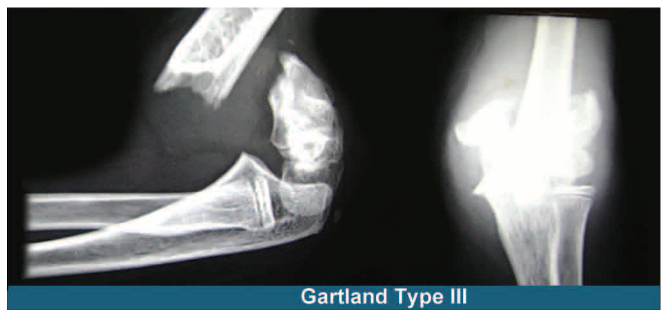


Figure 2.45

Anteroposterior and lateral view showing a badly displaced extension type of supracondylar fracture of the humerus. The chances of neurovascular complication is very high.

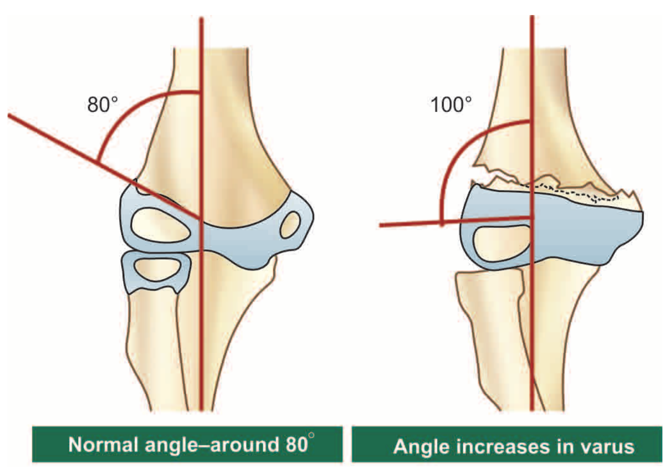


Figure 2.46

The Baumann's angle normal and abnormal.

Complications

The complications seen in supracondylar fractures are classified as:

a. Immediate
b. Late

Immediate

- Injury to brachial artery resulting in 'Volkmann's ischemia'
- Injury to median, ulnar and radial nerves

Late

- Volkmann's ischemic contracture
- Malunion. Results in cubitus varus and 'Gun stock deformity'
- Myositis ossificans
- Limitation of movements

Complications of Supracondylar Fracture and their Management

Volkmann's Ischemia

It is a compartment syndrome occurring in the extremities due to sudden loss of blood supply. It was first described by Dr Richard von Volkmann (1830–1889), a German doctor in a paper on "noninfective ischemic conditions of various fascial compartments in the extremities" (1881).

If the ischemia is left untreated, it leads to a classical contracture known as Volkmann's ischemic contracture.

Gartland Type-II and Type-III fractures are more prone to injure the brachial artery and median nerve, as both these structures are situated in the midline. Hence, when the distal fractured fragment gets displaced posteriorly and laterally, the proximal fragment pierces the brachialis muscle and injures the brachial artery (Fig. 2.47). Injury to the vessel is rare in Gartland type-I and when seen, it is due to compression by a hematoma.

The pathology of Volkmann's ischemia begins with tissue necrosis and a vicious cycle follows as shown in Figure 2.48. This ischemic phenomenon passes through three stages or phases. They are:

i. Stage of impending ischemia (also known as stage of threatened ishemic contracture) lasts for 24–48 hours
ii. Stage of established ischemia, lasts from 48 hours to 3 weeks
iii. Stage of established ischemic contracture, seen after 3 weeks

The treatment in each of these three stages is different. Hence, it is important to identify the stages of 'Volkmann's ischemic phenomenon' by examining the patient and identifying the signs.

Signs of impending ischemia: The five cardinal signs of impending ischemia are the 'Five P's'.

i. Pain
ii. Pallor
iii. Pulselessness
iv. Parasthesia
v. Paralysis

(There is a 6th 'P' that is increased pressure in the compartment which is an investigation and needs a manometer for measurement).

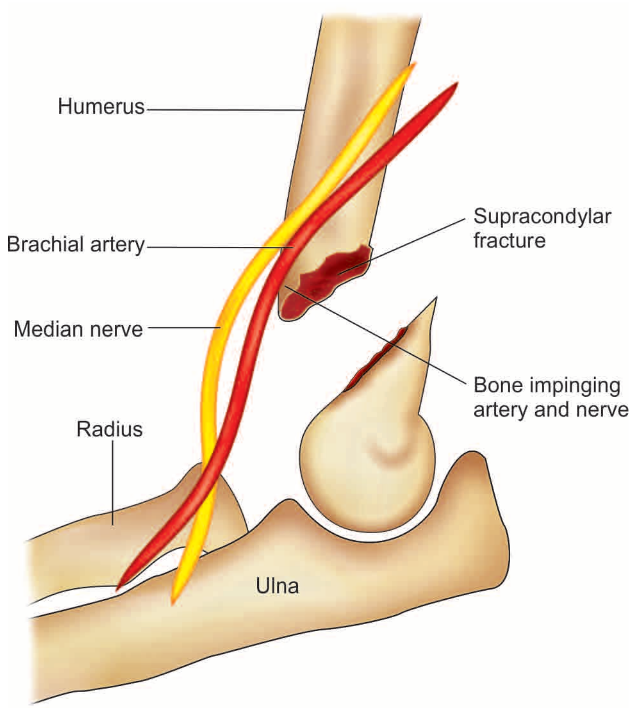


Figure 2.47
The midline disposition of brachial artery and median nerve and the likely mechanism of injury in a displaced supracondylar fracture.

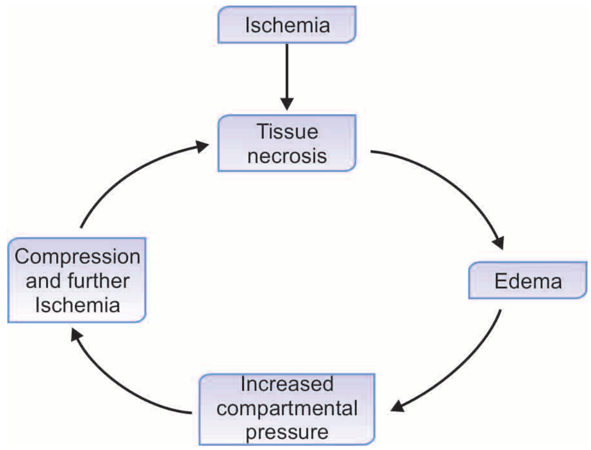


Figure 2.48
Vicious cycle of Volkmann's ischemia.

'Stretch pain' and 'Stretch sign': Seen in the stage of impending ischemia. With advancing ischemia, the flexor muscles become edematous. As a result, their muscle mass increases. In order to accommodate themselves comfortably within the tight fascial compartment of the forearm, they contract and reduce their muscle mass. This results in progressive flexion of the fingers.

The ischemic episode is painful. When the flexed fingers are stretched and extended passively by the examiner (thereby stretching the ischemic, contracted muscles) it results in excruciating pain. This is '*Stretch pain*'. The sign is called the '*Stretch sign*'.

Sign of established ischemia: The hand remains paralyzed with presence of only a flicker of movement or no movements at all. Sensory impairment also develops.

Note: *Paralysis occurring in 'Volkmann's ischemia' is mainly due to the ischemia of the muscles involved than the ischemia of the nerves.*

Sign of established contracture

'Volkmann's sign': It is a sign seen in 'Volkmann's ischemic contracture', and is described as "flexion of the wrist allows passive extension of the fingers and dorsiflexion does not".

Pathomechanics of 'Volkmann's sign': When there is ischemic contracture, the flexor muscles are fibrosed and shortened. Hence, dorsiflexion of the wrist does not allow passive extension of the fingers. Whereas, when the wrist is flexed these contracted and fibrosed flexor muscles are relaxed and there is relative lengthening of the fibrosed muscles. This allows passive extension of the fingers.

To summarize, 'Stretch pain' is the sign of impending ischemia, 'Paralyzed hand' is the sign of established ishchema and 'Volkmann's sign' is the sign of established contracture.

Treatment: It is an emergency. The treatment protocol depends on the stage at presentation.

The principle of treatment in the 1st stage or stage of impending ischemia: It is to restore the circulation back to normal and reverse the damage, thereby preventing progression of ischemia and development of contracture. It is a stepwise treatment protocol and at each step, attempt is made to restore and improve the circulation.

Methods employed to restore the circulation back to normal: The methods employed are as follows in a sequential order.

1. Split the tight plaster or cut the tight bandage, if any.
2. Emergency closed reduction of fracture with or without 'K-wire' fixation.
3. Open reduction and internal fixation of fracture using 'K-wires', along with exploration and repair of the damaged vessel.

Note: *After every step of treatment look for the return of pulse and capillary filling. At any point of time, if the circulation comes back, the next step in the sequential order to restore the circulation is not carried out.*

Vascular procedures employed to restore the circulation: The vascular procedures employed to restore the circulation, depend on the nature of the vascular injury which may be a spasm, a laceration, a cut vessel, or a crushed vessel (e.g. open fracture). Following are the different procedures employed.

1. Injection of 1% lignocaine or papaverine into the vessel wall—if only a spasm of the vessel is detected and there is no damage. This will relieve the spasm and restore the circulation.
2. Repair of the laceration, when a laceration is detected on exploration.
3. End-to-end anastomosis, in a cut vessel.
4. Resection and a vein graft, in a crushed vessel.
5. Thrombectomy of the distal vessel may have to be done during vascular repair using Fogarty's catheter.

Indicators for return of circulation: The indicators for return of circulation are:

Clinical

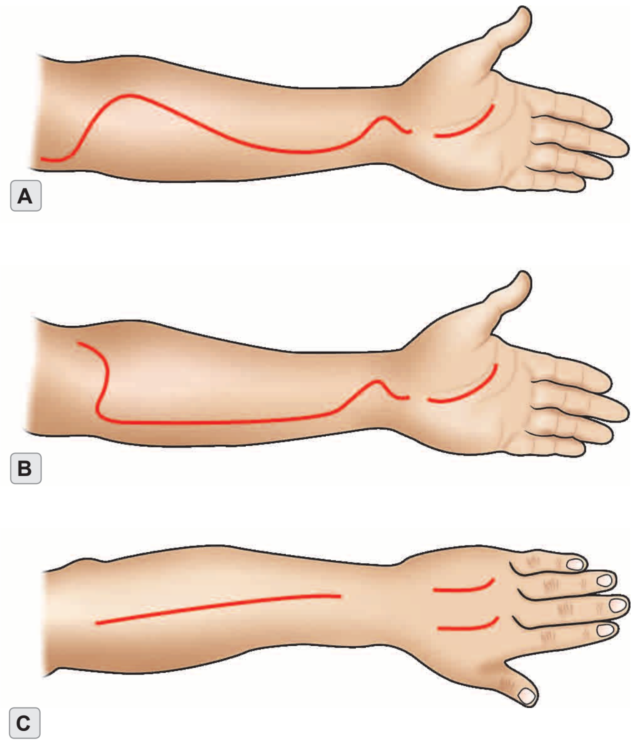
a. Return of pulse
b. Return of capillary filling

Confirmatory (employing gadgets)

a. Pulse oximeter showing a good saturation of oxygen above 90%.
b. Doppler probe picking up a good signal of blood flow.

Principle of treatment in the 2nd stage: The principle of treatment in the 2nd stage is to improve the circulation by decreasing the compartmental pressure.

Surgical procedure employed in the 2nd stage: The surgical procedure employed is a wide fasciotomy (Fig. 2.49A to C) which is carried out by dividing the tight deep fascia, dividing the aponeurosis and the septae, excising the dead and devitalized tissue and leaving the wound open either for a secondary closure by suturing or by skin grafting procedure. Nerves are released by neurolysis. Vessels and tendons are always protected. This procedure of decompression, is supposed to open up the collaterals, perfuse the remaining tissues and minimize the contracture. It is also essential to use an appropriate splint to support and protect the limb in order to minimize contracture.



Figures 2.49A to C
Different incisions used for decompression of forearm and hand compartment.

In a transition phase between the 1st and the 2nd stage, both vascular repair and fasciotomy may have to be done (i.e. vascular repair is followed by fasciotomy).

Principle of treament in the 3rd stage: The principle of treatment in the 3rd stage is to restore some useful function in a hand with ischemic contracture.

Restoration of useful function: This is accomplished by means of plastic and reconstructive procedures such as release of contractures, muscle-sliding procedures, lengthening of tendons, tendon transfers, shortening of the bones, fusion of joints, etc. as per the merit of the case, age of the patient and type of ischemic contracture.

Types of ischemic contracture:

a. Mild type: This involves only wrist flexors i.e. flexor carpi radialis and flexor carpi ulnaris (FCR and FCU).
b. Moderate type: This involves wrist flexors as well as finger flexors i.e, flexor digitorum superficialis, flexor digitorum profundus and flexor pollicis longus (FCR, FCU + FDS, FDP, FPL).
c. Severe type: This involves both flexor and extensor group of muscles.

The different methods employed in the managment of different stages of Volkmann's phenomenon are summarized in Flow chart 2.1.

Nerve Injuries

In extension type of supracondylar fracture, median nerve injury is most common, followed by the ulnar nerve and then the radial nerve.

In flexion type of supracondylar fracture, ulnar nerve injury is most common, followed by the median nerve and then the radial nerve.

Type of nerve injury: In closed fractures, it is either neuropraxia or axonotmesis. Hence, proper splinting followed by supervised physiotherapy, results in normal functional recovery (see chapter on peripheral nerve injury).

In open fractures, however, there is a possibility of a neurotmesis. In such cases, nerve repair is indicated, i.e. neurorrhaphy. Recovery depends on severity of neurotmesis and quality of repair (see chapter on peripheral nerve injury).

At times, there is a possibility of a nerve getting adhered in the callus or myositis mass. It needs release of a nerve and neurolysis. Recovery is slow and results are unpredictable.

Malunion

Malunion results in cubitus varus and 'Gun stock' deformity. Rarely cubitus valgus.

Note: *Malunion in cubitus varus with internal rotation results in a 'Gun stock' deformity (Fig. 2.50). Cubitus varus alone does not constitute a gun stock deformity.*

Definition of carrying angle, cubitus varus and cubitus valgus: The angle made by the long axis of the arm

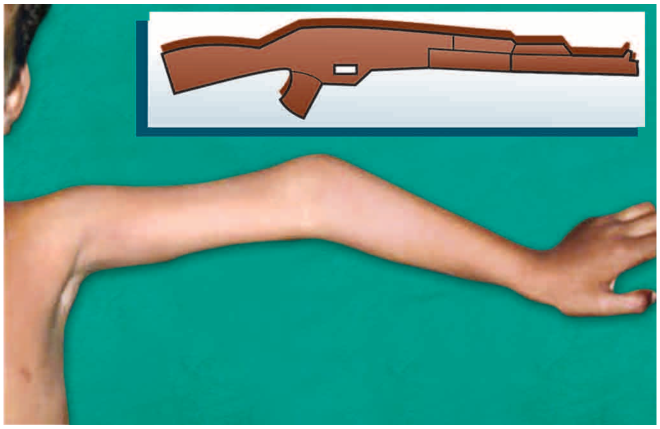
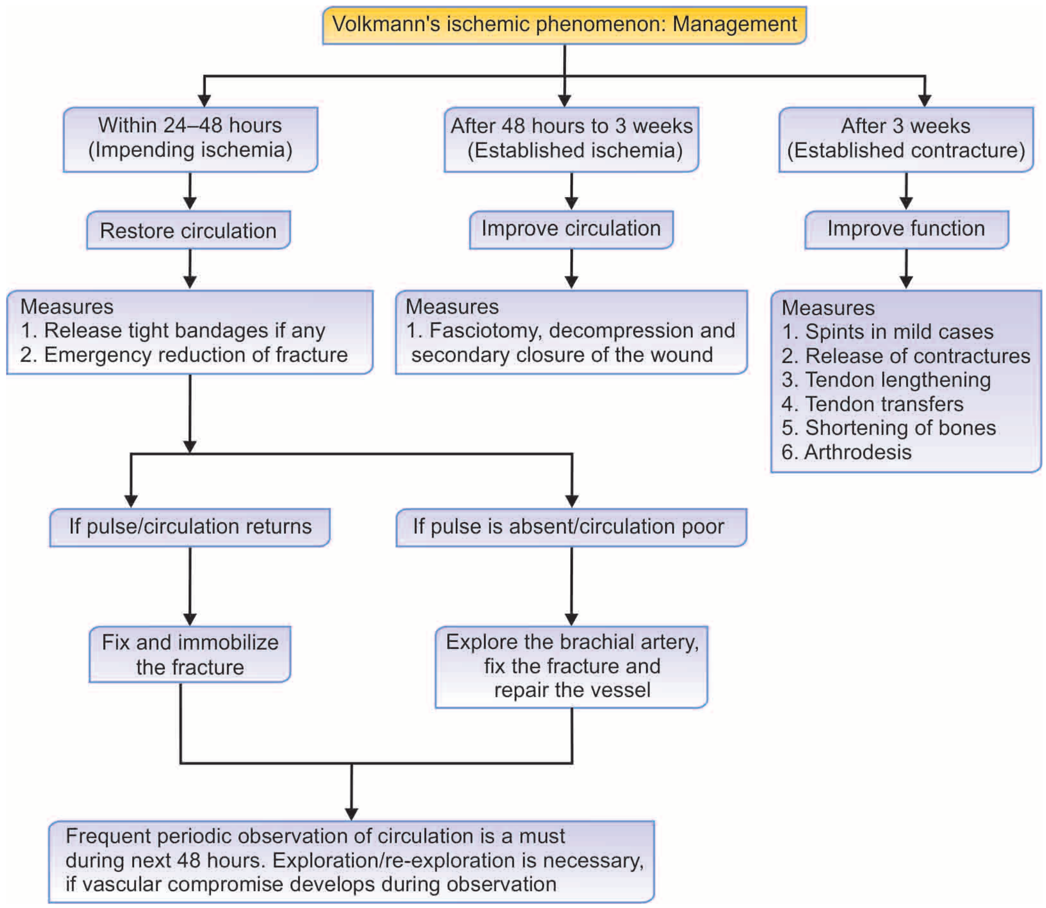


Figure 2.50
A classical 'Gun stock deformity' of a malunited supracondylar fracture.

Flow chart 2.1

Management of Volkmann's ischemic phenomenon in a supracondylar fracture.



with the long axis of the forearm in extended position of the elbow is known as 'Carrying angle'. Normal angle is 5–10° in males and 10–15° in females.

When this 'Carrying angle' reverses it is said that the elbow is in cubitus varus and when this 'Carrying angle' increases it is said that the elbow is in cubitus valgus.

Note: *If there is a flexion deformity at the elbow carrying angle cannot be measured. This angle is always measured with the elbow in extension.*

Surgical procedure for correction of deformity: The deformity is corrected by means of a corrective osteotomy. French osteotomy corrects both varus and internal rotation malalignment of a 'Gun stock' deformity. It is a lateral closed wedge, derotational type of osteotomy, fixed internally with 'fig of 8' stainless steel (SS) wires, wound round the screws which are passed proximal and distal to the site of osteotomy, prior to the osteotomy in a specific manner (Figs 2.51A to C).

Rare deformity of 'Cubitus Valgus' is corrected by medial closed wedge osteotomy.

Myositis Ossificans

It is an ossification or calcification occurring within a muscle following injury. Brachialis muscle is the commonly affected muscle. This complication impairs movement and causes stiffness of the elbow.

Treatment: When myositis is in acute and active phase no intervention should be attempted. Intervention in any form will aggravate the condition. When the acute phase subsides and the mass lies dormant, surgical removal followed by adequate gradual and active physiotherapeutic mobilization to relieve stiffness and regain movement is necessary.

Figures 2.51A to C

Classical procedure of French osteotomy done for a gun stock deformity in malunited supracondylar fracture. (A) Note the appropriately marked wedge of bone to be removed, (B and C) the placement of screws for closing the wedge and binding them with SS wire. The lower screw is placed anterior to the upper screw. Because of this, when the wedge is closed and the screws are aligned, along with correction of varus deformity the internal rotation deformity also gets corrected.

Revision Questions

General

Q. What is the age group for supracondylar fractures?
Q. What are the types of supracondylar fractures of the humerus?
Q. Which type of supracondylar fracture is common?
Q. What is the mechanism of injury?
Q. What is the nature of the fracture line?
Q. What is the importance of knowing the nature of the fracture line in a supracondylar fracture?
Q. What is the role of muscle triceps and brachialis in the treatment of supracondylar fracture?
Q. When do you do 'K-wire' fixation of a supracondylar fracture?

Complications

Q. What are the complications seen in supracondylar fracture?
Q. What is "Volkmann's ischemia" and ischemic contracture?
Q. What are the 3 stages of " Volkmann's ischemic phenomenon" ?
Q. What is importance of understanding the 3 stages?
Q. What are the cardinal signs of impending ischemia?
Q. What is 'Stretch pain and stretch sign'?
Q. What is 'Volkmann's sign'?
Q. What is the principle of treatment in the 1st stage of ischemia?
Q. What are the methods employed to restore the circulation back to normal at earliest possible moment?
Q. What are the vascular procedures employed to restore the circulation?
Q. What is the principle of treatment in the 2nd stage of ischemia?
Q. What is the surgical procedure indicated in the second stage of ischemia?
Q. What is the principle of treatment in the 3rd stage of ischemia?
Q. What are procedures indicated in the 3rd stage of ischemia for restoration of useful function?
Q. Name the nerves that may get injured in a supracondylar fracture.
Q. What is the nature of nerve involvement in a supracondylar fracture?
Q. What is the prognosis of nerve injury in a supracondylar fracture?
Q. What are the deformities seen in a malunited supracondylar fracture?
Q. What is 'Carrying angle' at the elbow?
Q. What is cubitus varus and cubitus valgus?
Q. How do you correct the deformity in malunited supracondylar fracture?
Q. What is myositis ossificans?
Q. Which muscle is commonly affected in myositis ossificans?
Q. What is the treatment of myositis ossificans?

Essay Questions

Q. What are the types of supracondylar fractures of the humerus? Discuss the mechanism of injury, diagnosis and treatment of supracondylar fracture. Enumerate the complications.

Q. What are the complications of supracondylar fracture? Discuss the management.

Q. What is Volkmann's ischemic phenomenon? Discuss the management of Volkmann's ischemia and contracture.

Q. What is gun stock deformity? Discuss the management of gun stock deformity.

Intercondylar Fracture

These are among the fractures seen at the lower end of the humerus and result following severe trauma. The fracture line in these fractures runs between the two condyles of the lower humerus and hence derives the name, intercondylar fracture. The line also extends in the supracondylar portion of the lower humerus either horizontally or obliquely giving the shape of alphabet 'T' or 'Y' to this fracture.

Mechanism of Injury

As a result of severe impact on the point of the elbow, the olecranon process is firmly driven upwards, splitting the condyles into two. Comminution is seen when the impact is severe.

Types

A. 'T' type—In this the humeral fracture line is transverse and the intercondylar fracture line is vertical resembling the letter T.

B. 'Y' type—In this the humeral fracture line on either side is obliquely placed to the vertical intercondylar fracture line resembling the letter Y.

Diagnosis

Proper X-rays are essential for clear definition of the injury. When there is bad comminution CT scan is indicated for assessment of the fracture.

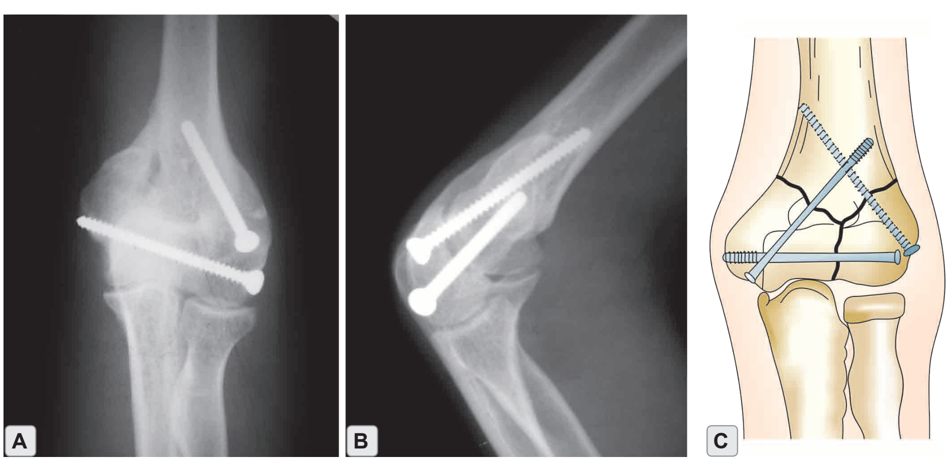
Treatment

Nonoperative: There is a limited role for nonoperative treatment. It is indicated only in patients who are not fit for surgery or the injury is so severe that the elbow is reduced to a bag of bones. In such cases, the elbow is initially splinted. After the initial swelling subsides, a hinge brace is given and movement of the elbow is encouraged.

Skeletal traction for a limited period is the other option available.

Operative: Surgical treatment definitely gives better results with respect to union and function.

Experience and skill of the surgeon has a direct bearing on the functional outcome. Surgery aims at restoring the medial and the lateral column. Though congruity of the articular surface is restored, limitation of movements of flexion and extension in terminal degrees is common. This is attributed to the severity of surrounding soft tissue injury. One-third tubular plates, Recon plates and screws (both cortical and cancellous) are the implants used to stabilize these fractures (Figs 2.52A to C). External fixators are used in open fractures.



Figures 2.52A to C

(A) Intercondylar fracture—AP view; (B) Intercondylar fracture—lateral view; (C) Fixation of a 'Y' type of noncomminuted intercondylar fracture using cancellous and cortical screws.

Total elbow replacement is an option which may be considered in elderly, when the elbow is reduced to a bag of bones.

Complications

Early

1. Injury to the brachial artery.
2. Injury to the median/ulnar/radial nerve.

Late

1. Limitation of joint movements.
2. Myositis ossificans.

FRACTURES IN THE ARM AND AROUND THE SHOULDER

Fracture Shaft of the Humerus

General Information

This fracture is also known as a diaphyseal fracture of the humerus. The shaft of the humerus bone is an area, extending from the upper border of pectoralis major muscle in the shoulder region to supracondylar ridge at the elbow. The shape of the bone is cylindrical in its proximal half but changes to triangular or prismatic in its distal half. The two intermuscular septae, namely, the medial and the lateral divide the area into two compartments, i.e. anterior and posterior. The anterior compartment contains biceps brachii, coracobrachialis and brachialis muscles; brachial vessels, namely the brachial artery and vein as well as the median, musculocutaneous and ulnar nerves. The posterior compartment contains the triceps and the radial nerve. Although these fractures are inherently unstable, nonoperative method is the treatment of choice especially in the young except when definite indications for surgery exist.

Mechanisms of Injury of the Fracture Shaft Humerus (Figs 2.53A to C)

Indirect mechanism:

Is by means of a fall on an outstretched hand.

a. A bending force produces a transverse fracture.
b. A torsional force produces a spiral fracture.
c. A combination of both bending and torsional force produces a comminuted fracture with butterfly fragments.

Direct mechanism: Is by means of a blow on to the arm which results in a shattered displaced fracture of the shaft of the humerus, e.g. assault by a stick, a high velocity injury, etc.

Figures 2.53A to C

Transverse, spiral and comminuted fracture of the humerus at different levels: (A) Transverse fracture in the middle 1/3rd; (B) Spiral fracture in the upper 1/3rd; (C) Comminuted fracture in the lower 1/3rd.

Treatment

A. *Nonoperative:* The nonoperative methods of treatment of this fracture are:
   a. Closed reduction and maintenance in a 'U' slab or a cast (Fig. 2.54A).
   b. Maintaining the fracture reduction in a 'Hanging cast' (Fig. 2.54B).

B. *Operative (Figs 2.55A and B):*

   Indications
   1. Noncompliance.
   2. Unacceptable reduction (failure of closed reduction).
   3. Displaced, comminuted or segmental fracture.
   4. Multiple fractures (polytrauma, ipsilateral ulna or radius fracture).
   5. Open fractures.
   6. Fractures associated with neurovascular injury.
   7. Fractures with intra-articular extension.

Implants used for surgery:
   i. Various plates and screws, e.g. LC DCP, LCP.
   ii. Intramedullary nails e.g. interlocking nails, flexible nails like Enders, AO titanium nails, etc.

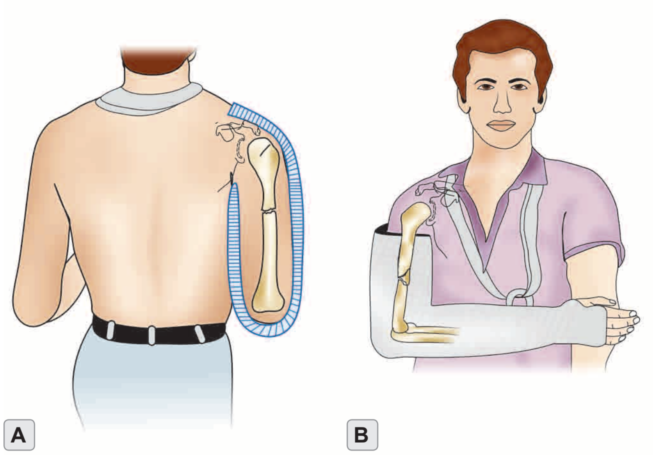
External fixators are used in open fractures.

Holstien-Lewis fracture: This specific type of injury was described by Arthur Holstein and Gwilym B Lewis. Hence the name is derived.

It is a fracture of the lower 1/3rd of the humerus with entrapment of radial nerve resulting in radial nerve palsy. The radial nerve pierces the lateral intermuscular septum and lies anteriorly at the elbow between brachialis and brachioradialis. In a fracture of the lower 1/3rd of the humerus, when the distal fragment gets displaced proximally and laterally, it carries the intermuscular septum and the piercing radial nerve along with it. This may lead to entrapment of the radial nerve and sometimes even laceration of the nerve causing damage and palsy. Primary open reduction is the treatment of choice for this injury. The radial nerve should be explored at the time of fracture fixation.

Complications

1. Radial nerve injury (incomplete or complete)
2. Delayed union
3. Nonunion



Figures 2.54A and B

Different nonoperative treatment methods of fracture shaft of humerus: (A) 'U' slab; (B) Hanging cast.

Figures 2.55A and B

(A) A delayed union of fracture shaft of humerus; (B) Internal fixation using LC DCP and cancellous bone grafting from the iliac crest.

Treatment of Complications

Radial nerve injury: The radial nerve gets injured because of its close relation to the bone in the radial groove. The type of nerve injury is either neuropraxia or axonotmesis. Patient develops wrist drop which generally recovers on its own over a period of time. Drug therapy splints and physiotherapeutic measures help in the recovery (Refer chapter on peripheral nerve injuries). Surgical exploration is rarely needed (Fig. 2.56).

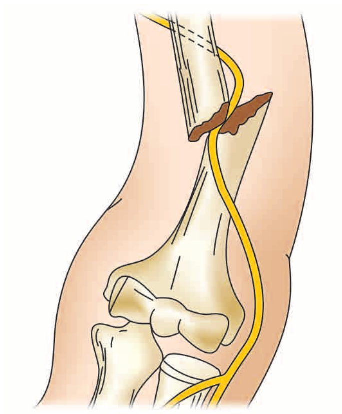


Figure 2.56

Radial nerve entrapment in a fracture shaft humerus as a result of its close relation to the shaft of the humerus in the radial groove.

Delayed union and nonunion: The delayed union and nonunion of fracture shaft of humerus is treated by means of internal fixation and cancellous bone grafting.

Revision Questions

Q. What are the mechanisms of injury in fracture shaft humerus?

Q. Discuss the treatment of fracture shaft humerus.

Q. Enumerate the complications of fracture shaft humerus and discuss their management.

Fractures of the Proximal Humerus

General Information

In the year 460BC, Hippocrates documented a case of fracture neck humerus first and treated it by using traction. Codman, in 1934 divided the proximal humerus into four parts based on epiphyseal lines. The head (the articulating surface with anatomical neck), the greater tuberosity, the lesser tuberosity and the shaft. The surgical neck is distal to both the tuberosities and it is that portion of bone between the tuberosities and the shaft. According to Codman, fractures of the proximal humerus produce a combination of the four segments.

The blood supply is from the branches of the axillary artery, running in a distal to proximal direction. Hence, fracture of the anatomical neck may result in loss of blood supply to the head of the humerus and avascular necrosis of the head of the humerus.

Shoulder is an important ball and socket type of a joint linking the upper extremity to the thorax. Because of an arc of movement taking place at the shoulder joint, an individual is able to perform explosive activities involving power, e.g. throwing an object, as well as activities that are refined such as playing a violin. So, fracture of the proximal humerus can cause severe disability in the upper extremity by limiting the function of the shoulder.

The fracture is commonly seen in elderly females (M:F ratio 1:2) as a result of low velocity injury.

In young it occurs as a result of high velocity injury.

Mechanisms of Injury

1. Common mechanism—by a fall on an outstretched hand from a standing height.
2. Other mechanism—a direct blow on to the proximal humerus.

Classification

The commonly accepted classification is ‘ Neer’s classification’ (Fig. 2.57).

This system of classification includes four parts (segments):

- The head of the humerus
- The greater tuberosity
- The lesser tuberosity
- The shaft of the humerus.

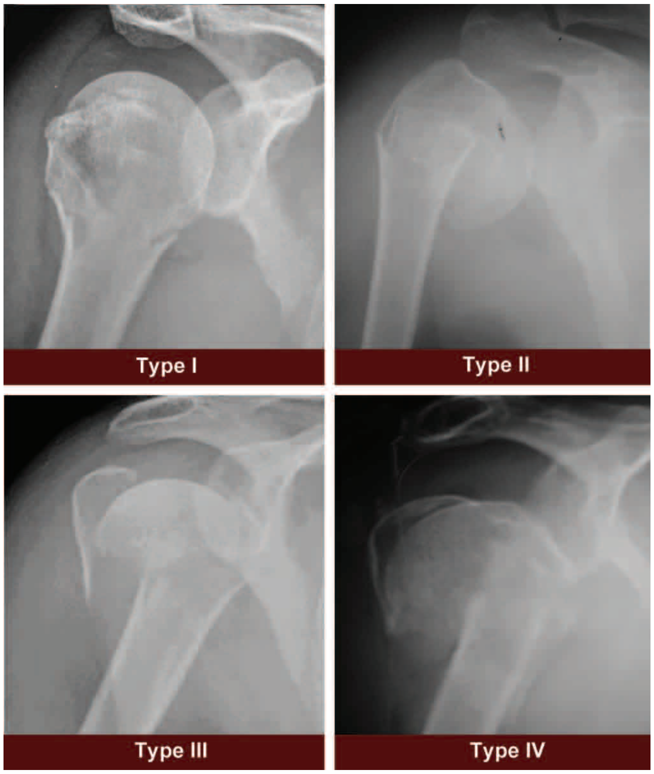


Figure 2.57

Different types of surgical neck fractures based on Neer’s classification (Refer text).

According to Neer, a fracture is displaced when there is more than 1 cm of displacement and >45° of angulation of any one fragment with respect to the others.

Displacements occur because of the muscle pull.

a. The supraspinatus and the infraspinatus pull the greater tuberosity superiorly.
b. The *subscapularis* pulls the *lesser tuberosity medially.*
c. The *pectoralis major adducts the shaft medially.*

Type-I: It is an undisplaced fracture or displacement less than 1 cm.
Type-II: The 'Two-part fracture' involves any two of the 4 parts and include 1 fragment that is displaced.
Type-III: The 'Three-part fracture' includes a displaced fracture of the surgical neck in addition to either a displaced greater tuberosity or lesser tuberosity fracture.
Type-IV: The 'Four-part fracture' includes displaced fracture of the surgical neck and both tuberosities.

Treatment

A. *Nonoperative:* Undisplaced or minimally displaced fractures are managed nonoperatively and immobilized in plaster slab or by a *Velpeau* bandage and strapping.
B. *Operative:* Displaced fractures and fractures that pose difficulty for closed manipulation are managed by surgical intervention.

Methods employed: The surgical methods employed are:

1. Closed reduction and percutaneous fixation.
2. Open reduction and internal fixation using plates and screws, e.g. 'T' plates, proximal humerus locking plates (Fig. 2.58).
3. Minimally invasive percutaneous plate osteosynthesis.
4. Primary arthroplasty (3 part and 4 part fractures and fractures involving head especially in middle aged and elderly).

Complications

1. Malunion
2. Nonunion
3. Avascular necrosis of the head of the humerus

Treatment of Complications

Malunion: The scapulothoracic movement compensates to a certain extent, the limitation of movement that occurs at the glenohumeral joint secondary to malunion. Corrective osteotomy is rarely indicated.

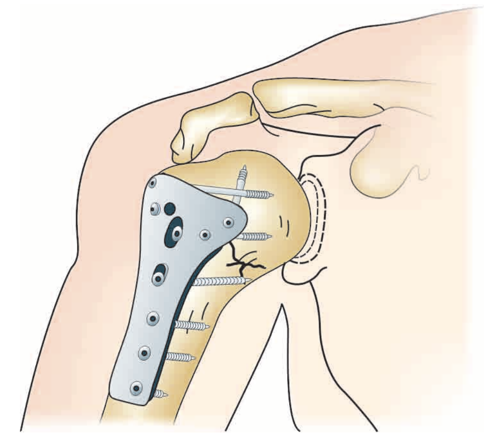


Figure 2.58
Method of fixation of proximal humeral fracture with T-plate (Proximal humerus LCP) and screws.

Nonunion and AVN are managed by shoulder arthroplasty using prosthesis, e.g. Neer's prosthesis.

Restoration of useful shoulder movement is the principle involved in these surgeries.

Revision Questions

Q. What are the mechanisms of injury in fracture surgical neck of humerus?
Q. Which is the commonly accepted classification of fracture neck humerus?
Q. How do you manage this fracture?
Q. What are the surgical methods employed to treat this fracture?
Q. What are the complications seen?
Q. How are these complications managed?

Fracture of the Clavicle

General Information
Also known as collar bone, clavicle is the only bone which connects shoulder girdle to the trunk (connects the breast bone, i.e. sternum to shoulder blade, i.e. scapula).

It is a curved subcutaneous bone which changes its shape from round and somewhat cylindrical medially, to flat and quadrilateral laterally. It protects the underlying vessels, lungs and brachial plexus. It undergoes membranous ossification and is the only horizontal bone in the human body.

Though this fracture is seen in all age groups, it is most commonly seen in neonates (following a difficult delivery) and in children.

Mechanisms of Injury

1. Fall on an outstretched hand
2. Fall on the point of a shoulder
3. A direct blow on to the clavicle

Displacing forces are shown in Figure 2.59.

Common Site and Classification (Fig. 2.60)

- 80% occur in the middle 1/3rd (Class A)
- 15% occur in the lateral or distal 1/3rd (Class B)
- 5% occur in the medial or proximal 1/3rd (Class C)

Note: *Class B is further classified as:*

I. Nondisplaced with intact supporting ligaments.
II. Displaced because of coracoclavicular ligament rupture and pull of the sternocleidomastoid muscle.
III. Articular fracture involving the acromioclavicular joint.

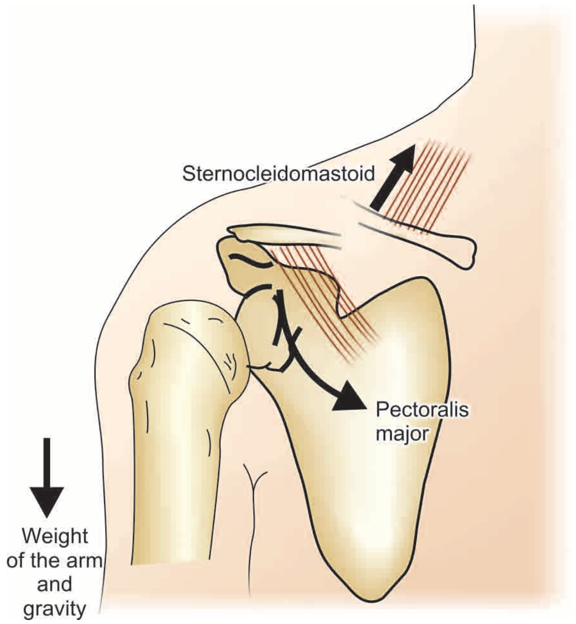


Figure 2.59

Various displacing forces in a fracture clavicle.

Treatment

In infants and children, fracture invariably heals with simple strapping or even with an arm sling immobilization.

In adults, treatment depends on the Class of the fracture (Figs 2.61A to C).

Class A fractures—These fractures are treated commonly with immobilization in a Figure of 8 bandage and a sling. Displaced fractures which are amenable for closed reduction, are reduced before application of figure of '8' bandage. Badly displaced fractures which do not get satisfactorily reduced are treated by ORIF using clavicle LCP.

Class B fractures—Type I needs only supportive strapping and a sling for the arm and fracture heals without any problem.

Type II and III need surgical methods for reduction and stabilization, e.g. transarticular 'K'-wire, repair of coracoclavicular ligament, etc.

Class C displaced fractures—These are treated by surgical reduction.

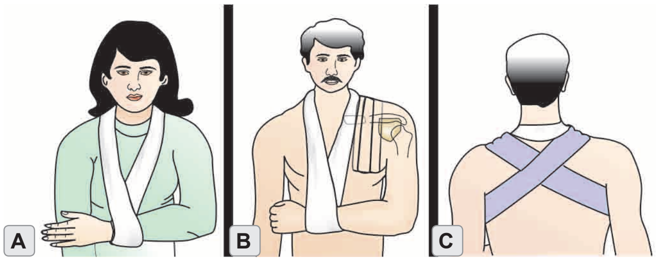


Figure 2.61A to C

Methods of conservative treatment of fractures clavicle: (A) Collar and cuff sling; (B) Strapping and sling suspension; (C) Figure of 8 bandaging.

Figure 2.60

Different types of clavicular fracture Class A fracture, i.e. in the middle 1/3rd: Class B fracture, i.e. in the lateral end or distal 1/3rd and Class C fracture, i.e. in the medial 1/3rd.

Complications and Treatment (In Brief)

1. Life-threatening and limb threatening complications may be associated with fracture clavicle, e.g. hemothorax, pneumothorax, hemopneumothorax, injury to subclavian vessels, injury to brachial plexus, etc. These are surgical emergencies and need specialized care.
2. Delayed union and nonunion are managed only if symptomatic. Internal fixation and bone grafting is done in such cases. If asymptomatic and no disability is observed they are left alone.
3. Malunion is generally left alone.

Revision Questions

Q. What are the mechanisms of injury in fracture clavicle?

Q. What is the classification of fracture clavicle?

Q. What is the treatment?

Q. What are complications associated with fracture clavicle? How do you manage them?

Essay Question

Q. Discuss the mechanism of injury, diagnosis and management of fracture clavicle. Enumerate the complications.

Fractures of the Scapula

Mechanisms of Injury

1. A direct blow on to the scapula, e.g. assault, fall of a heavy object (Fig. 2.62).
2. Axial loading through the glenohumeral joint.

Sites of the Fracture (Figs 2.63A and B)

In the order of frequency the fracture sites are as follows:

a. Fracture of the body
b. Fracture of the neck
c. Fracture of the glenoid
d. Fracture of the acromion
e. Fracture of the coracoid

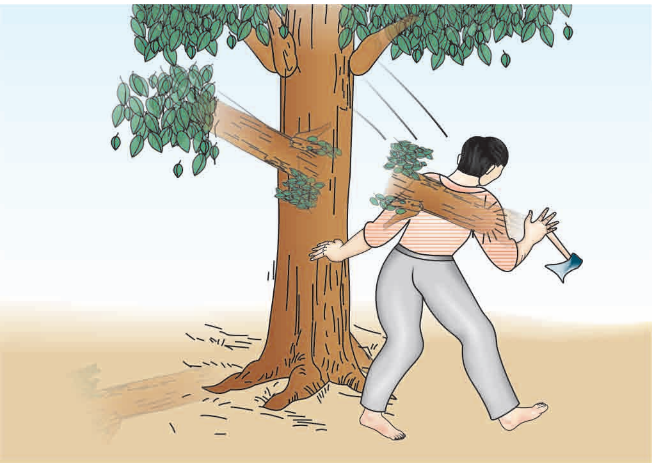


Figure 2.62

Most common mode of injury causing scapular fracture is a direct impact on the scapula. The diagram shows a direct impact by a branch of a tree falling upon the shoulder blade.

Other Injuries Associated with Fracture Scapula and their Incidence

The incidence of associated injuries is around 80–90%. The following injuries are commonly seen:

1. Pulmonary contusion and pneumothorax (23%)
2. Clavicle fracture resulting in 'Floating shoulder' (23%)
3. Anterior or posterior dislocation of the shoulder
4. Brachial plexus and axillary artery injury
5. Rib fracture

Note: *Scapular body fractures heal without any problem in about 6 weeks. A simple immobilization in a sling is sufficient. Similarly, a fracture of the acromion process of the scapula, is treated nonoperatively unless it causes compression of the rotator cuff.*

Classification of the Scapular Neck Fractures

The scapular neck fractures are classified as follows:

Type I—Nondisplaced and nonangulated

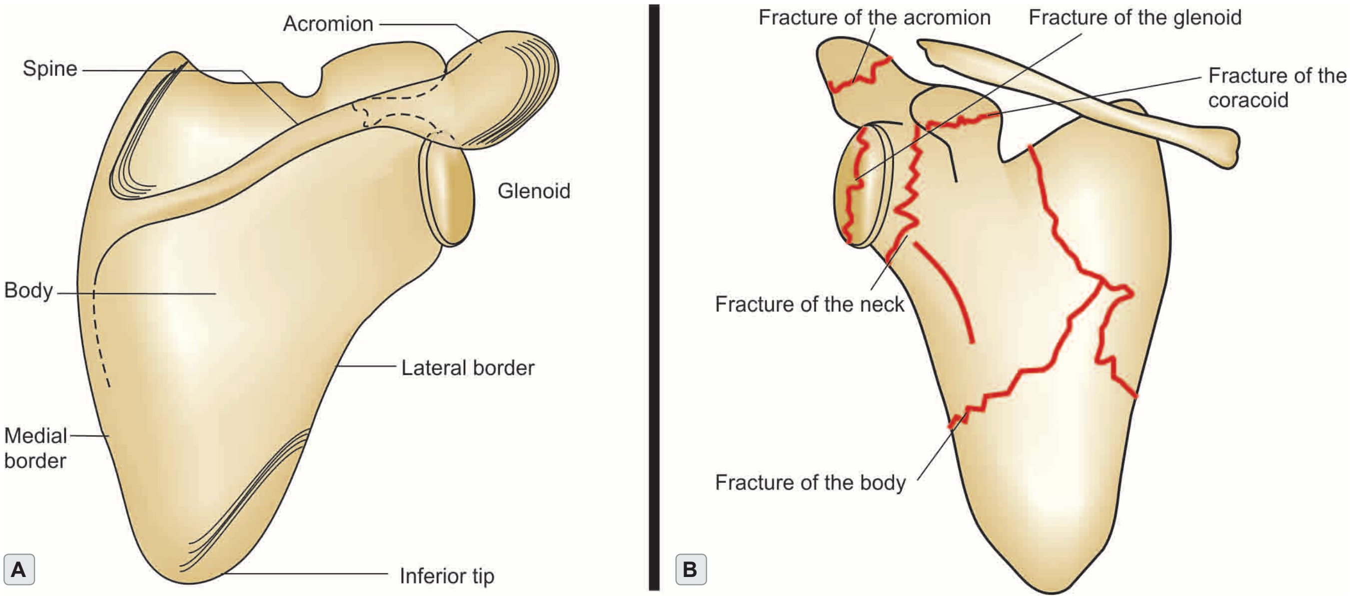
Type IIa—Shortened/displaced >1 cm

Type IIb—Angulated > 45°

Treatment of Scapular Neck Fractures

Type I—are treated nonoperatively and yield good functional results.

Type IIa and IIb fractures need surgical treatment as they are displaced and angulated.



Figures 2.63A and B
(A) Bony anatomical features of scapula; (B) Different types of fracture scapula.

Classification of the Fractures of the Glenoid

Based on Ideberg classification classified as:

Type I—Anterior avulsion fractures
Type II—Transverse, inferior glenoid fractures
Type III—Transverse superior glenoid fractures
Type IV—Transverse fractures through the body
Type V—A combination of Type II and Type IV

Indications for surgery in glenoid fractures: Those fractures which are likely to cause instability are definite indicators of surgery. They are:

1. Rim fractures with > 1 cm displacement/involving > 25% of the articular surface.
2. Intra-articular fractures with subluxation of the head of the humerus.
3. Glenoid fossa fractures which are displaced > 5 mm.

Surgical approaches used: Anterior fractures are exposed by means of anterior exposure to the shoulder and posterior fractures are exposed by the posterior exposure.

Implants that are used to fix these fractures: The various implants used to fix these fractures are 3.5 mm cortical screws, 4.0 mm cancellous screws (for lag effect) and 1/3rd tubular plate (for buttress effect).

Revision Questions

Q. What are the sites of the fracture in the scapula?
Q. What are the injuries associated with fracture scapula and their incidence?
Q. How do you classify scapular neck fractures?
Q. What is the treatment of scapular neck fracture?
Q. How do you classify the fractures of the glenoid?
Q. What are the indications for surgery in glenoid fractures?
Q. What are the surgical approaches used?
Q. What are the implants that are used to fix these fractures?

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3 Ligament Injuries and Fractures in the Lower Limb

Introduction, general information, mechanism of injury, diagnosis, treatment and complications are dealt with as follows:

- *Fractures of the foot*
- *Injuries around the ankle*
- *Injuries around the knee joint*
- *Fracture of the shaft of the femur*
- *Fractures around the hip*

FRACTURES OF THE FOOT

Introduction

The foot has a very important role in locomotion. It is subjected to constant stress of weight bearing. Loss or impairment of anatomy of the foot following injury may thus lead to serious disability and malfunction. Hence, a subspecialty of ***Foot and ankle surgery*** has evolved and meticulous foot and ankle surgery is being performed these days.

The foot is generally divided into (Fig. 3.1):

A. Hindfoot consisting of calcaneus and talus.
B. Midfoot consisting of navicular, cuboid and the three cuneiforms.
C. Forefoot consisting of metatarsals and phalanges.

Hindfoot

The articulation between the talus and the calcaneus is known as subtalar joint. Inversion and eversion movements take place at this joint.

The talus articulates with the ankle mortise formed by the lower end of tibia and fibula. This forms the ankle joint. Dorsiflexion and plantar flexion movements take place at the ankle joint.

Midfoot

The tarsal bones of the midfoot have multiple articulations. Movements at these articulations occur in a very unique manner, as a unit. When inversion/eversion takes place at the subtalar joint, the tarsal bones lock themselves and move together only to get unlocked when a movement occurs in the opposite direction (eversion/inversion).

It is this, effective, constant and multidirectional adjustment of the tarsus, helps the foot to adapt

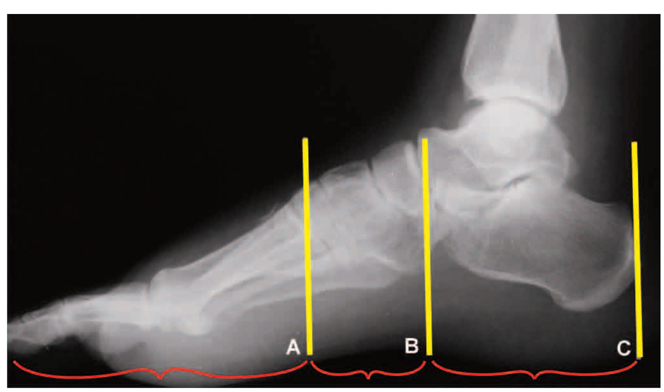


Figure 3.1

Lateral radiograph of a normal foot showing: The forefoot (A); The midfoot (B); The hindfoot (C).

itself to any terrain which results in a smooth propulsion. Thus, loss or impairment of this mechanism following injury to the tarsal bones/ articulation, results in severe disability.

The articulation between the hindfoot and the mid-foot is known as midtarsal joint formed by talonavicular and calcaneocuboid joints. It is also known as ***Chopart's joint*** named after ***Francois Chopart*** (1743–1795) who performed amputations at this level—***Chopart's amputation.*** The procedure removes almost all the insertions of tendons around the ankle and renders the ankle joint unstable.

The articulation between the midfoot and forefoot is known as ***Lisfranc's joint*** named after ***Jacques Lisfranc*** a French surgeon in Napoleon's army. An amputation at this level is known as ***Lisfranc's amputation*** as he performed amputations at this level for frostbite and gangrene, on soldiers at the Russian front.

Forefoot

The articulations between the metatarsals and phalanges are more firm and regular and do not allow multidirectional adjustments. These joints are more involved in transmission of body weight during push off phase of gait cycle. Hence, any disturbance in anatomy following injury will affect the push off phase of gait cycle (See chapter on gait).

The important injuries occurring in the foot are:

1. Chopart's injury
2. Lisfranc's injury
3. Jones' fracture
4. Pseudo-Jones' fracture
5. Talus fracture
6. Calcaneus fracture

Chopart's Injury

Definition

It is a fracture dislocation involving the talonavicular and the calcaneocuboid joint (Midfoot).

Mechanisms of Injury

Common mechanism (80%): Is a severe twisting injury on a plantar flexed and inverted foot causing medial and upward displacement of distal fragments.

Rare mechanism: Is an 'Eversion injury' which causes lateral displacement of distal fragments.

In both the mechanisms, the talus always remains in the ankle mortise.

Fractures associated with Chopart's injury

1. Calcaneus fractures
2. Talus fractures
3. Navicular fractures

Management

Prompt and early accurate reduction along with stabilization is the treatment of choice. Hence, nonoperative methods have a limited role to play. They are useful only in minor grades of injury.

Prognosis

Poor prognosis is seen in those cases which are associated with severe soft tissue injury, fracture of other bones of the foot, persistent malalignment after treatment and in those which present late.

Lisfranc's Injury

Definition

This is a fracture or a dislocation or a fracture dislocation occurring at the junction between the tarsal bones of the midfoot and the metatarsal bones of the forefoot named after ***Jacques Lisfranc*** a French surgeon in Napoleon's army.

Note: *Lisfranc described this injury in soldiers who fell down from their horses with their foot caught in the stirrup (a fall without foot getting released from the stirrup).*

Mechanism of Injury

Mechanism involves severe plantar flexion of the foot, e.g. stepping into a small hole, sports-related injuries, motor vehicle accidents, falling from a height etc.

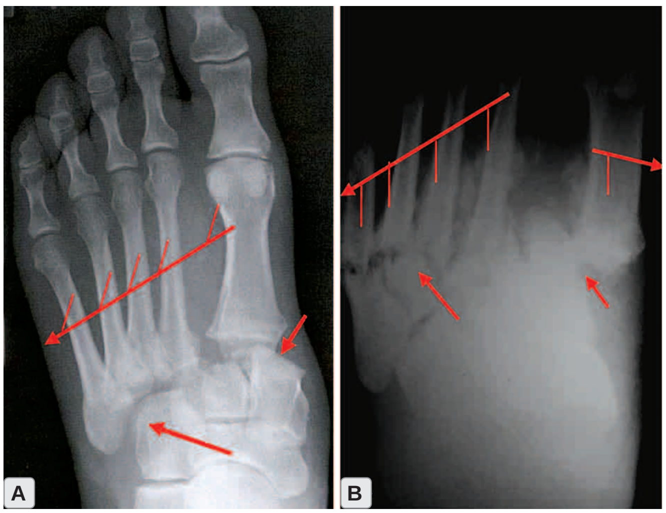
Lisfranc ligament diagonally connects the 1st (medial) cuneiform with the base of the 2nd metatarsal. If it remains intact at the time of injury, it exerts a traction force on the bone which depending on the direction of pull, causes, either an avulsion of the lateral border of the 1st (medial) cuneiform or an avulsion of the base or medial border of the 2nd metatarsal. If it tears, these fractures do not occur.

Types

Two basic types are described.

Homolateral (Fig. 3.2A)

- All the metatarsals are displaced to the same side.
- Usually 2nd to 5th metatarsals are involved which displace laterally as a unit.
- May involve all 5 metatarsals.
- More common than divergent.



Figures 3.2A and B

Radiographs of the foot showing (A) Homolateral and (B) Divergent types of Lisfranc's injury. Note the direction of displacements of the 1st metatarsal and the 2nd, 3rd, 4th and 5th metatarsals.

Divergent (Fig. 3.2B)

- Usually involves medial displacement of the 1st metatarsal and lateral displacement of 2nd-5th metatarsals.
- Occasionally medial displacement of only the 1st metatarsal may be seen.
- May be associated with a fracture of the 1st cuneiform.
- Usually more severe than homolateral.

Note: *Rarely, isolated injury involving one or two metatarsals which get displaced from others, is seen.*

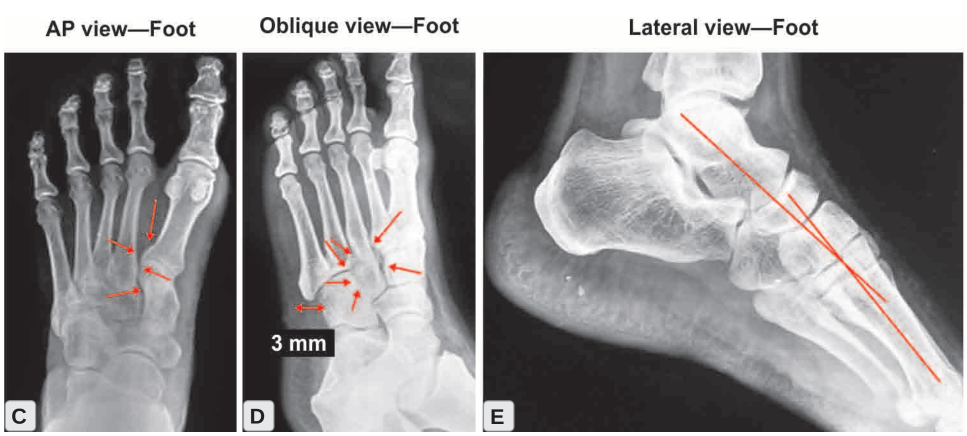
Fractures associated with Lisfranc dislocations

- Base of 2nd metatarsal fracture.
- Cuneiform fracture.
- Fractures of shafts of metatarsals.
- Dislocations of the 1st (medial) and 2nd (middle) intercuneiform joint and cuneonavicular joint.
- Fractures of the navicular bone.

Imaging (Figs 3.2C to E)

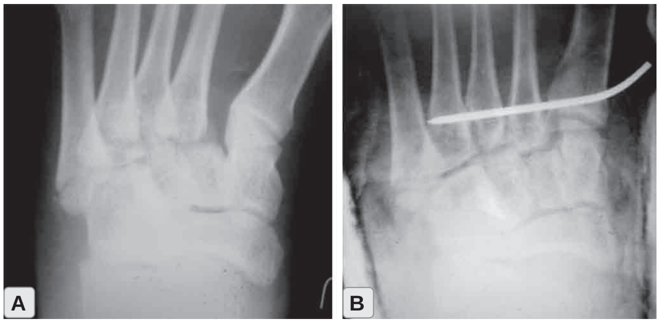
Conventional radiographs are usually sufficient to demonstrate the injury. Normal alignment of the cuneiforms and the bases of the metatarsals are as follows:

- Lateral border of 1st metatarsal is aligned with lateral border of 1st (medial) cuneiform in anteroposterior (AP) view.
- Medial border of 2nd metatarsal is aligned with medial border of 2nd (intermediate or middle) cuneiform in AP view.



Figures 3.2C to E

Radiograph of a normal foot showing (C) AP view of the foot. (D) Oblique view of the foot. (E) Lateral view of the foot (Refer text for details).



Figures 3.3A and B

Successful reduction and stabilization of a Lisfranc's injury—Homolateral type by (B) closed reduction and pinning. In this, the findings are fracture base of 2nd metatarsal, fracture base of 5th metatarsal—both are avulsion fractures and dislocation of 2nd to 5th metatarsals which are displaced laterally.

- Medial and lateral borders of the 3rd (lateral) cuneiform is aligned with medial and lateral borders of 3rd metatarsal in oblique view.
- Medial border of 4th metatarsal is aligned with medial border of cuboid in oblique view.
- Lateral margin of the 5th metatarsal may project lateral to cuboid by as much as 3 mm in oblique view.
- In lateral view, a line drawn along the long axis of talus intersects a line drawn along the long axis of the 1st metatarsal.

Any loss of this pattern of alignment is an indicator of this injury.

Management

Restoration of anatomical alignment at the earliest is the key for successful result. Hence, closed methods of reduction/fixation and immobilization have a role to play only in less severe forms of injury. Severe forms are always treated by open reduction and internal fixation (Figs 3.3A and B).

Complications

a. Compartment syndrome involving the foot compartment (immediate).
b. Deformities of the foot due to persistent malalignment resulting in chronic pain (early) and degenerative arthritis involving tarsal joints (late).

Prognosis

Severe soft tissue injury, persistent malalignment, associated fractures and late presentation results in poor prognosis.

Note: *Instability on weight bearing can occur with ligament injuries alone, without a fracture or a dislocation.*

Revision Questions

Q. What is Chopart's and Lisfranc's joint? Why it is named so?
Q. What is Chopart's and Lisfranc's amputation?
Q. What is Chopart's and Lisfranc's injury?
Q. What is the mechanism of Chopart's and Lisfranc's injury?
Q. What is Lisfranc's ligament? What are the types of Lisfranc's injury?
Q. What are the fractures associated with Chopart's and Lisfranc's injury?
Q. How do you manage Lisfranc's and Chopart's injury?
Q. What are the complications of Chopart's and Lisfranc's injury?
Q. What is the prognosis of Chopart's and Lisfranc's injury?

Fracture of the Fifth Metatarsal Bone

Fracture involving proximal part of the fifth metatarsal is the most common injury among metatarsal fractures. The distal and the mid shaft fractures of the 5th metatarsal are not common.

Types:
A. Jones' fracture.
B. Pseudo-Jones' fracture (Tennis fracture).

Jones' Fracture

This injury was first described in 1902 by Sir Robert Jones in an article titled "Fractures of the Base of the Fifth Metatarsal Bone by Indirect Violence." He himself is said to have suffered from this injury (fracture) after dancing.

Definition

It is a transverse fracture occurring at base of the fifth metatarsal 1.5–3.0 cm distal to the tuberosity, either at the metaphyseo-diaphyseal or proximal diaphyseal region (Fig. 3.4).

Figure 3.4

Radiograph of a Jones' fracture.

Figure 3.5

Pseudo-Jones' fracture.

Diagnosis

1. Pain in the region of the fifth metatarsal after an indirect violence.
2. Difficulty in walking.
3. X-ray confirming the diagnosis.

Treatment

Always treated by a non-weight bearing cast for the fear of becoming displaced and causing nonunion at a later period. Nonunion may need bone grafting.

In athletes and in displaced fractures, surgical stabilization is considered.

Pseudo-Jones' Fracture

Definition

It is an avulsion fracture occurring at the base of the fifth metatarsal as a result of the pull of peroneus brevis tendon (Fig. 3.5).

Treatment

Heals well with a compression bandage or a walking cast in about 3 weeks without complications.

Note: *Mechanism of injury—is almost similar both for Jones' and Pseudo-Jones' fractures. It is a lateral ankle strain with sudden inversion of the foot which causes these fractures. Depending on the area that is subjected to maximum stress, fractures occur in different regions of the fifth metatarsal i.e. from the base to the diaphysis.*

Fracture of the Talus

General Information

Talus also known as 'Astragalus', is a unique bone which has no muscle or tendon attachments and held in place mainly by bony and ligamentous support. Superiorly, it articulates with the inferior articular surface of the tibia; inferiorly with the superior articular surface of the calcaneus; medially with the medial malleolus; laterally with the lateral malleolus and anteriorly with the navicular.

Hence, it plays an important role in the function of the ankle and the foot by being responsible for 90% of the movement occurring at the ankle and the foot.

History

Sir Astley Cooper (1832) gave a first hand account of a case of dislocated talus which he treated. (Mr Downes 20th July 1820, who fell down from his horse......)

Anderson (1919) gave the name **'Aviator's Astragalus'** because he found these injuries occurring in Aviators when they crashed with their machines. The foot resting on the rudder bar took the impact of extreme dorsiflexion thereby causing injuries of the talus. Namely, fractures, dislocations and fracture-dislocations.

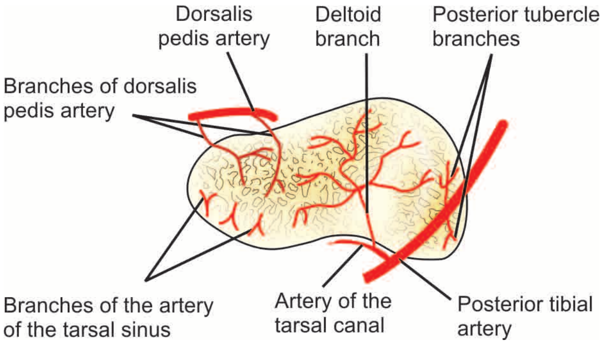


Figure 3.6

Blood supply to the talus. Note that the important supply is through the artery of the tarsal canal which anastomoses with the artery of the sinus tarsi.

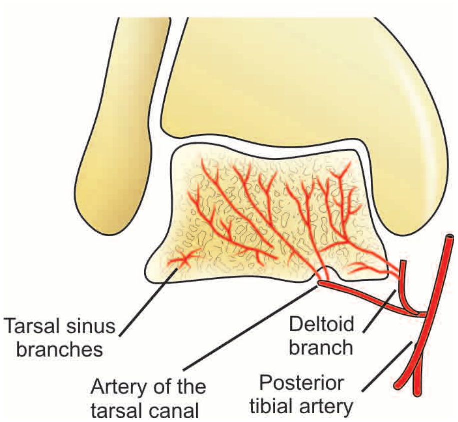


Figure 3.7

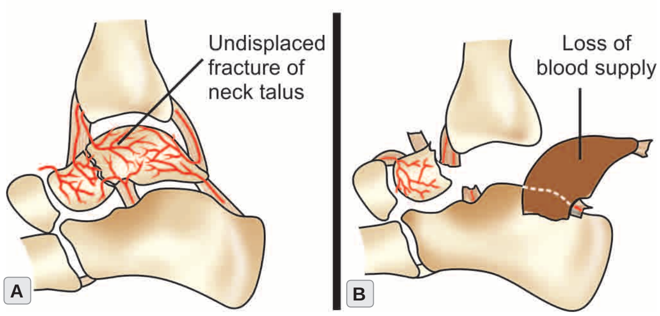
Blood supply of the talus in coronal plane.

Blood Supply (Figs 3.6 and 3.7)

GL Mulfinger and J Trueta have described in detail the blood supply of the talus (J Bone Joint Surgery 1970;52B.).

The blood supply to the talus is derived from three main arteries of the leg i.e. the anterior tibial, the posterior tibial and the peroneal artery through a *periosteal network* and through *two discrete vessels* as follows.

1. Posterior tibial artery—through calcaneal branches
2. Anterior tibial artery—through anterolateral/anteromedial malleolar branches or through medial tarsal artery
3. Peroneal artery—through perforating branches
4. Artery of the tarsal canal, a branch of posterior tibial artery
5. Artery of the sinus tarsi, a branch of dorsalis pedis artery



Figures 3.8A and B

(A) Fracture of the neck of the talus alone does not disturb the blood supply without displacement of the body; (B) When the body is displaced the blood supply is lost.

Note:

Two plexus are formed by anastomosis.

i. In the region of the posterior tubercle.

ii. In the region of the sinus tarsi.

The calcaneal branches of posterior tibial artery form a plexus in the region of posterior tubercle while the anterolateral malleolar branches of anterior tibial artery along with branches from dorsalis pedis artery form a plexus in the region of sinus tarsi. Thus, 'vascular slings' are formed all around the talus due to anastomosis.

An undisplaced fracture of the neck disturbs the blood supply the least. Hence, the incidence of AVN is low (Intact 'vascular slings').

A displaced fracture neck of the talus, disturbs the blood supply to the body and causes avascular necrosis. Similarly, a fracture of the body of the talus impairs the blood supply to the body and causes avascular necrosis (Damage to 'vascular slings') (Figs 3.8A and B).

Mechanism of Injury

Talar neck fractures: Extreme dorsiflexion of the ankle or direct axial loading causes rupture of the posterior capsule and impaction of the talar neck against the distal tibia resulting in a vertical fracture of the neck. Subsequently, one of two things can happen. Either the foot subluxates forwards with body of the talus adopting a position of equinus because of the recoil or if the dorsiflexion continues the body of the talus dislocates and is pushed backwards out of the ankle mortise. In the second scenario, the body of the talus goes on to lie between the medial malleolus and Achilles tendon with the fracture surface facing upwards and laterally (Ankle mortise has been described under ankle injuries).

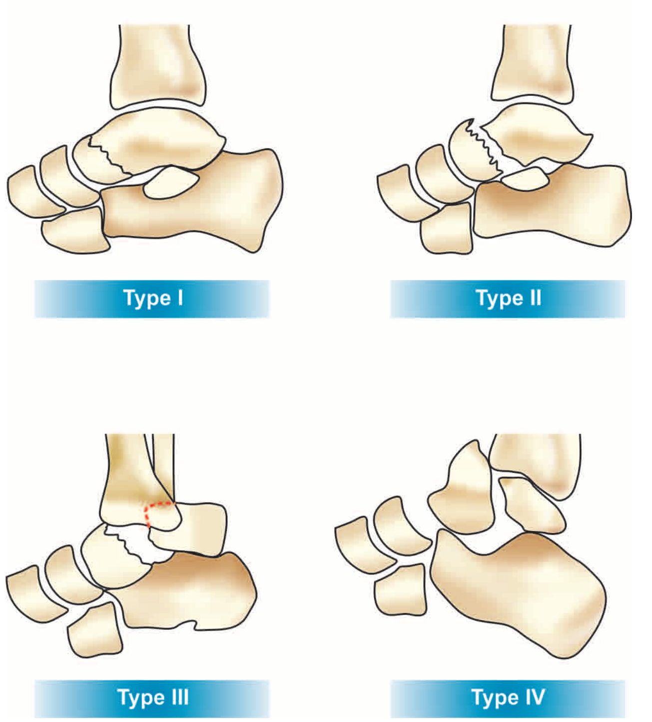


Figure 3.9

Different types of fracture of talus based on Hawkins' classification modified by Canale and Kelly.

Figure 3.10

Radiographs of different fractures based on Hawkins' classification and modification by Canale and Kelly. *Type I*: Undisplaced fracture. *Type II*: Displaced fracture with subtalar subluxation/dislocation. *Type III*: Displaced fracture with both subtalar and ankle subluxation/dislocation. *Type IV*: Displaced fracture with subtalar, ankle and talonavicular dislocation.

Classification of the Fractures/Dislocations and Fracture—Dislocations of the Talus

Following injuries are seen in the talus:

A. Talar neck fracture
B. Talar body fracture
C. Talar head fracture (rare)
D. Subtalar dislocation
E. Total talar dislocation

Classification of talar neck fracture

Based on Hawkins' classification (1970) it is classified as follows (Figs 3.9 and 3.10):

I. Undisplaced vertical fracture.
II. Displaced fracture with subtalar joint subluxation/ dislocation.
III. Displaced fracture with both subtalar and ankle dislocation.

Canale and Kelly, proposed a IV Group.

IV. Displaced fracture with subtalar, ankle and talonavicular dislocation.

Classification of talar body fractures

Based on Sneppen classification (1977) they are classified as follows:

I. Osteochondral dome fractures
II. Coronal, sagittal, horizontal shear fractures
III. Posterior process fractures
IV. Lateral process fractures (Snowboarder's fracture)
V. Crush fractures

Talar head fractures are rare and generally associated with talonavicular dislocation/subluxation.

Complications

a. Osteochondral fracture may cause loose body inside the joint which results in persistent pain and limitation of movement.
b. Nonunion in fracture neck of the talus.
c. Avascular necrosis of the body of the talus.
d. Degenerative arthritis of the ankle.

Figure 3.11A and B

AP and lateral view of ankle arthrodesis done for an old fracture talus with nonunion and AVN of the body. Note that the calcaneus is not plantigrade. The ankle is in 5 degrees of dorsiflexion. This will result in calcaneocavus deformity of the foot over a period of time when the patient starts weight-bearing.

Hawkins' sign:
This is a sign seen at the end of 6-8 weeks which indicates presence of good vascularity. This can be clearly visualized in a mortise view as a subchondral radiolucent band in the dome of the talus.

Treatment

A. *Nonoperative:* Fractures which are undisplaced/displaced less than 1 mm are immobilized in plaster cast.

B. *Operative:*

Internal fixation: Displaced fractures such as Types II, III and IV neck fractures; coronal, sagittal and horizontal shear fractures of the body are openly reduced and internally fixed.

Arthroscopy: Arthroscopic procedures are done for removal of loose body, management of osteochondral lesions and in early degenerative arthritis of ankle.

Arthrodesis: Indicated in secondary complication of nonunion, avascular necrosis and degenerative arthritis (Figs 3.11A and B).

Total joint arthroplasty: Indicated in secondary degenerative arthritis (without AVN).

Talectomy or Astragalectomy: Rarely done as it renders the ankle highly unstable.

Essay Questions

Q. Describe the blood supply of talus. Discuss the mechanism of injury, diagnosis and management of fracture neck of talus and its complications.

Q. Classify fractures of the talus. Discuss the management of the fractures of the talus. Enumerate their complications.

Calcaneus Fracture

Also known as " Lover's fracture"and "Don Juan fracture".

Functional Importance of the Calcaneus

a. Acts as a lever arm for gastrosoleus complex.
b. Transmits the body weight.
c. Supports and maintains the lateral column of the foot.

Hence, when its anatomy is not restored after a fracture the above functions get impaired and the gait is affected.

Surgical Anatomy

The calcaneus has two articular surfaces.

i. Anterior
ii. Superior.

The anterior articular surface is saddle shaped and articulates with the cuboid bone.

The superior articular surface has three articular facets.

1. The concave anterior facet.
2. The concave middle facet.
3. The convex posterior facet.

The anterior facet articulates with the anterior talar facet and is supported by the beak of the calcaneus. The middle facet lies over sustentaculum tali. Between the middle and the posterior facet is the calcaneal groove/sulcus.

Those fractures which involve the articular surfaces produce complex articular surface irregularities. Hence they need to be managed accurately in order to reduce the functional disability after recovery.

Figures 3.12A to C

Different types of calcaneal fractures based on Essex-Lopresti classification: (A) Extra-articular type; (B) Tongue, type; (C) Joint depression type.

Mechanisms of Injury

a. *Axial loading and shear:* Occurs when a person falls from a height and lands on the heel. The talus is driven down onto the calcaneus causing an articular surface fracture thereby disturbing the subtalar joint anatomy.
b. *Direct impact by blunt force:* Results in extra-articular fractures of the plantar tuberosity and the body.
c. *Twisting injuries:* Results in avulsion fractures of the calcaneus because of the pull of tendons and ligaments. For example, posterior tuberosity fracture due to the pull of tendo-Achilles.

Note: *When a person presents with calcaneus fracture, there is always a need to examine the pelvis, spine and the other limb, especially when there is a history of fall from a height or a high velocity injury. This is to rule out more serious injuries such as fracture of the pelvis and the spine. Also, it is very important to note whether there is developing compartmental syndrome involving the central compartment of the foot***.**

Classifications of Fracture Calcaneum

Essex-Lopresti classification (Figs 3.12 and 3.13):

a. Extra-articular
b. Intra-articular
   – Tongue type
   – Joint depression type

When there is an impact of axial loading, the calcaneum is subjected to two deforming forces which are transmitted through the talus.

a. Shearing force
b. Compression force

The pathomechanics following such an impact is explained below.

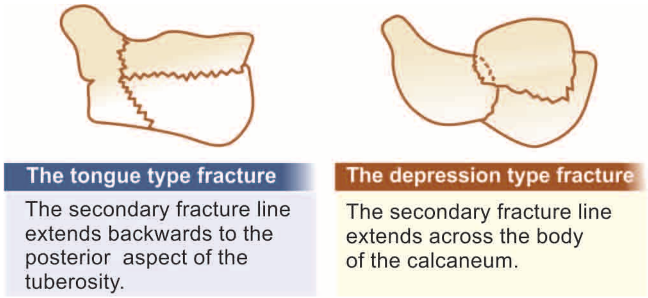


Figure 3.13

Diagrammatic representation of Essex-Lopresti classification of fracture involving the joint.

Pathomechanics of intra-articular fracture: When the calcaneus is subjected to axial loading the sharp edge of the lateral process of talus is driven into the 'crucial angle of Gissane' of the calcaneus (see Fig. 3.16B). Because of this, a shear fracture of the body develops with the fracture line extending from the lateral cortex to sustentaculum tali medially. This passes through the medial 1/3 or 1/2 of the posterior facet of the calcaneum.

If the force continues, a compression stress generated causes a secondary fracture. This fracture may be a tongue type or joint depression type.

Sander's classification: Developed in 1992, based on coronal and axial CT scans of the calcaneus. This classification is useful for deciding the treatment as well as reporting the results. This system defines four types of fractures (Fig. 3.14):

Type 1—Undisplaced fractures
Type 2—2 part split fractures
Type 3—Depressed fractures and/or 3 part split fractures
Type 4—4 part/comminuted fractures

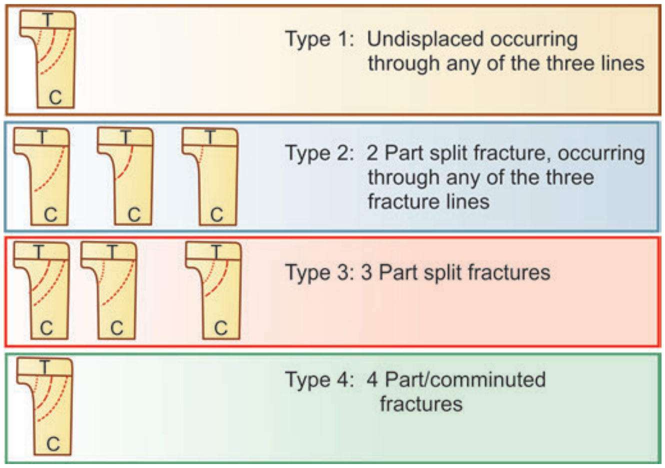


Figure 3.14

Diagrammatic representation of calcaneal fractures in coronal plane based on Sander's classification. *T—talus; C—calcaneus

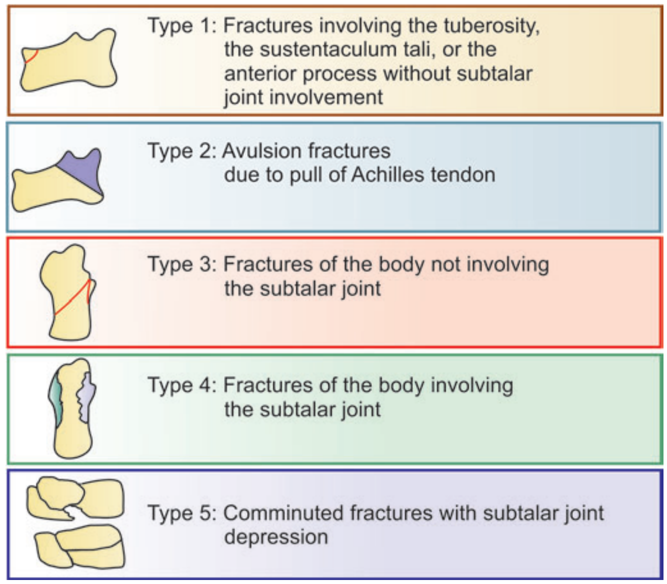


Figure 3.15

Diagrammatic representation of calcaneal fractures based on Rowe classification.

Accordingly, patients with type 1 injuries do well with nonoperative treatment; patients with type 2 and 3 injuries may be treated effectively with open reduction and internal fixation; type 4 injuries defy operative reduction.

Rowe classification: Based on this classification the fracture is classified into five types as follows (Fig. 3.15).

Type 1a—Tuberosity fracture medial or lateral

Type 1b—Fracture of the sustentaculum tali

Type 1c— Fracture of the anterior process of the calcaneus

Type 2a—Avulsion fractures involving posterior calcaneus (Beak fractures)

Type 2b—Avulsion fracture involving the insertion of the tendo-Achilles

Type 3— Oblique fracture of the body not involving the subtalar joint

Type 4—Body fracture involving the subtalar joint

Type 5—Body fracture with subtalar joint depression and comminution

Radiology in Fracture Calcaneum

Following views are necessary for a detailed evaluation:

- Lateral view—for assessment of 'Bohler's tuber angle' and 'crucial angle of Gissane' (Figs 3.16A and B).
- Axial view (Harris view)—for assessment of subtalar joint involvement.
- Anteroposterior view—for assessment of calcaneo-cuboid joint and avulsion fractures at the anterior and lateral part of the calcaneus.
- Oblique view (Anthonsen view and Borden view)—for assessment of articular extension of fractures.
- CT scan in complex and comminuted fractures for detailed and 3D evaluation.

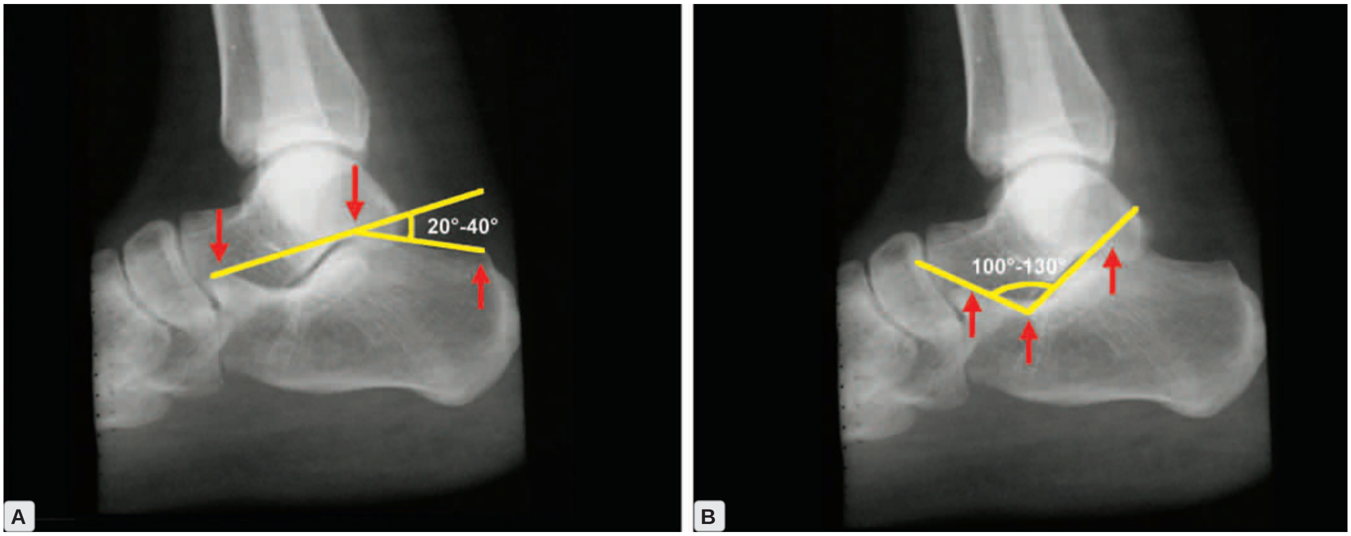
Treatment

Aim of treatment of fracture calcaneum is to maintain the 'crucial angle of Gissane' and 'Bohler's joint tuber angle' and maintain the anatomy of calcaneocuboid articulation (when indicated).

Undisplaced fractures as well as fractures which do not involve the articular surface (extra-articular fractures) are treated nonoperatively by application of well padded bandages, plasters slabs, casts and splints depending on severity of injury and immobilized for a period of 2–6 weeks. This is followed by gradual mobilization.

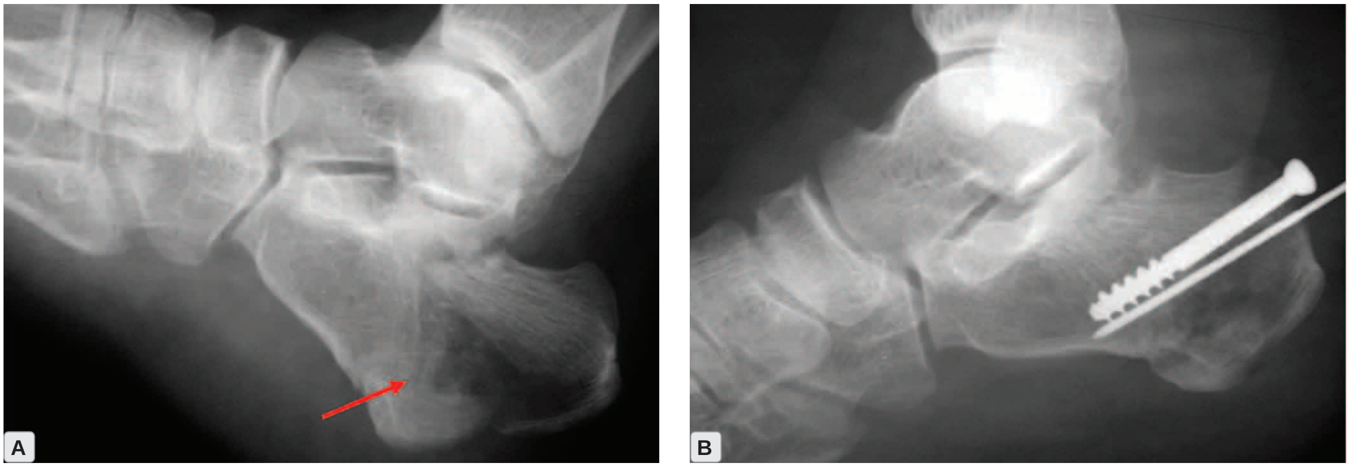
Displaced fractures as well as fractures which involve the articular surface and subtalar joint (intra-articular fractures) are treated by closed/open anatomical reduction and internal fixation using K-wires, cancellous screws or special plates, e.g. H-plates.

Avulsion fractures are treated by closed/open reduction and internal fixation (Figs 3.17A and B).



Figures 3.16A and B

(A) Normal 'Bohler's joint tuber angle' is 20–40°. It is an angle formed by two intersecting lines drawn between the superior margin of the posterior facet to the superior margin of the anterior process, extended proximally and another line drawn from the same point on the superior margin of the posterior facet to the superior margin of the tuberosity. The angle decreases or at times reverses in a posterior facet collapse; (B) Normal 'crucial angle of Gissane' is 100–130°. It is formed by the lines drawn between the lateral margin of the posterior facet and the anterior process. It increases in posterior facet depression.



Figures 3.17A and B

Radiographs of an extra-articular fracture of the calcaneum fixed by closed reduction and internal fixation. The technique of fixation was as follows. A pin was driven through the fractured bone. With the pin as the lever the fracture was manipulated and reduced with the foot in plantar flexion. Then the pin was driven further through the fracture site and the fracture was firmly transfixed. Next the cancellous screw was passed to achieve firm fixation. The transfixing wire may be kept *in situ* or may be removed.

Severely comminuted and complex fractures are generally treated by arthrodesis of the subtalar joint (Arthrodesis is a secondary procedure. Rarely it is done as a primary procedure).

Revision Questions

- Q. Discuss briefly the surgical anatomy and the functions of the calcaneum.
- Q. What is the mechanism of injury in fracture calcaneum?
- Q. Classify fracture calcaneum.
- Q. Draw the 'Bohler's joint tuber angle' and 'crucial angle of Gissane' and discuss their clinical importance.
- Q. How will you proceed with the investigations in a case of fracture calcaneum?

Essay Question

- Q. Classify fracture calcaneum. Discuss the mechanism of injury, diagnosis and management of fracture calcaneum. Briefly discuss the prognosis.

INJURIES AROUND THE ANKLE

Injuries around the ankle fall into two groups:

A. Ligament injuries
B. Bony injuries

Ligament Injuries

The common ligaments injured are the lateral ligament, the medial or the deltoid ligament and the syndesmotic ligament (the ligament binding the lower tibia and the fibula, i.e. inferior tibiofibular syndesmosis). These injuries are classified into three grades.

- Grade 1—Stretching of the ligaments
- Grade 2—Partial tear of the ligaments
- Grade 3—Complete tear of the ligaments

Management

Complete and severe tears require surgical repair. Less severe, strains and sprains heal well with adequate immobilization. Physiotherapy and gradual mobilization is always essential for a good recovery.

Bony Injuries of the Ankle (Pott's Fracture)

Pott's Fracture

All bony injuries of the ankle are grouped under one eponym, i.e. **'Pott's fracture'** because, it was Sir Percivall Pott (1714–1788) who first made an attempt to describe the various injury patterns occurring at the ankle.

It was also thought that Sir Percivall Pott had this fracture, after he fell off his horse in the year 1756. But, the fact was that Sir Percivall Pott actually had a compound fracture of the femur.

Hence, the term is considered to be misnomer. Sir Percivall Pott, neither described these injuries completely nor suffered this fracture.

Definition

Pott's fracture is defined as a uni, bi or trimalleolar fracture with or without subluxation or dislocation of the ankle joint.

Points to Remember

Ankle joint is a beautiful **hinge joint** which allows only the **movement of *dorsiflexion and plantar flexion.*** This movement takes place in a mortise known as ankle mortise formed by medial, lateral and posterior malleolus (posterior 1/3rd of the inferior articular surface of the lower tibia is known as posterior malleolus). The ligaments that support the mortise are the deltoid ligament medially, the lateral ligament laterally and the syndesmotic ligament superiorly.

Any other movement occurring in the ankle joint other than dorsiflexion and plantar flexion, is abnormal and tends to injure the supporting bony and ligamentous structures, leading to deformation of the joint. Failure patterns thus seen are several, because the ankle joint is amenable for a variety of abnormal stresses. These failure patterns are similar and are reproducible by a specific type of stress. This has been accurately described by Lauge-Hansen in this 'Genetic Classification' which he derived after experiments on cadavers. He used a wrench and subjected the cadaveric ankle to a variety of stresses and documented the results. It is worth noting that a particular type of stress produced a particular failure pattern and these patterns of failure were reproducible.

Lauge-Hansen' Genetic Classification

In this classification, the position of the foot at the time of impact as well as the direction of deforming force is

taken into account and the failure patterns are recorded. Accordingly, the injuries are classified as follows:

1. Abduction injuries (pronation abduction)
2. Adduction injuries (supination-adduction)
3. Pronation external rotation injuries
4. Supination external rotation injuries
5. Vertical compression injuries
6. Unclassifiable

Note: *All the terms used in the classification refer to the (abnormal) movements of the talus in the ankle mortise. If the ligament is stronger than the bone, the bone fractures and if the bone is stronger than the ligament, the ligament ruptures. It is also essential to remember certain 'Keywords' in order to understand the mechanism of ankle injuries.*

Keywords
Traction force—Pulling
Compression force—Direct impact
Partial/incomplete failure—Action of the force is incomplete
Total/complete failure—Action of the force is complete
Failure pattern—End result
Diastasis—Separation

Pathomechanics of Injuries

Abduction injuries: In these, the talus abducts in the ankle mortise. Abduction occurs in the vertical axis of the talus. The foot is in pronation when this occurs. Hence, the term pronation-abduction injury is used. When the talus abducts, it exerts a traction force on the medial structures and a compression force on the lateral structures.

Complete failure pattern: Traction on the medial structures causes either a transverse pull-off fracture of the medial malleolus OR a rupture of the deltoid ligament (depending on the strength of the ligament and the bone). Thus results in failure of the medial structures. (Medial support)—Stage 1.

The talus which is now free of its medial tether abducts further and hits the lateral malleolus thereby exerting compression force causing oblique or comminuted fracture of the lateral malleolus—Stage 2 (Fig. 3.18).

Adduction injuries: (Mechanism is exactly opposite to that of the abduction injuries). In these, the talus adducts in the ankle mortise. Adduction occurs in the vertical axis of the talus. The foot is in supination when this occurs. Hence, the term supination-adduction injury is used. When a talus adducts, it exerts a traction force on the lateral structures and a compression force on the medial structures.

Complete failure pattern: Traction on the lateral structures causes either a transverse pull-off fracture of the lateral malleolus OR a lateral ligament rupture (depending on the strength of the ligament and the bone). This results in failure of the lateral structures. (Lateral support)—Stage 1.

The talus which is now free of its lateral tether adducts further and hits the medial malleolus thereby exerting compression force causing oblique fracture of the medial malleolus—Stage 2 (Fig. 3.19).

Complete failure pattern in both abduction and adduction injuries is a bimalleolar fracture dislocation/subluxation of the ankle joint (except when ligament rupture occurs instead of a malleolar fracture), the dislocation/subluxation being lateral in abduction injuries and medial in adduction injuries.

Incomplete failure pattern presents either with isolated ligament rupture or unimalleolar fracture with or without subluxation of the ankle joint.

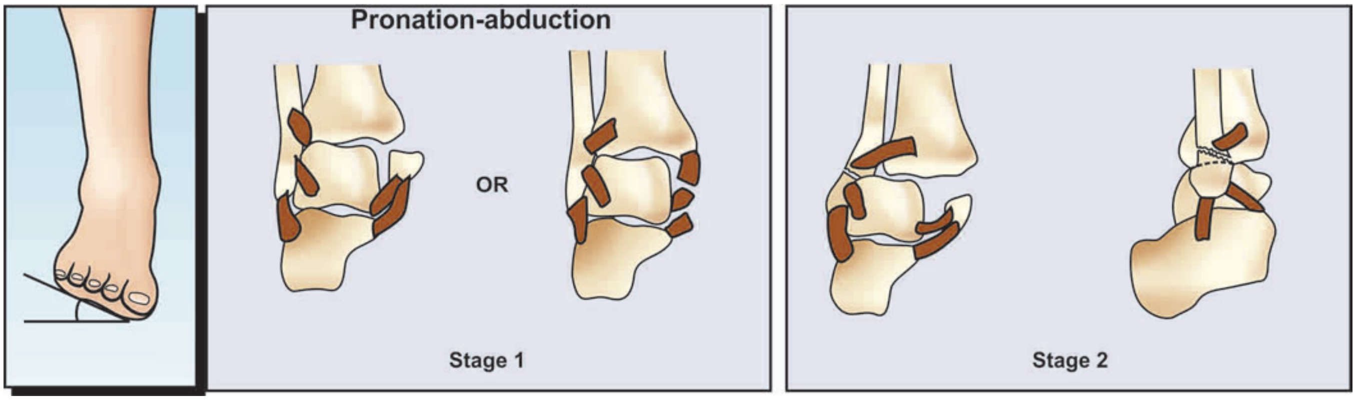


Figure 3.18
Diagrammatic representation of the sequence of events in abduction/pronation-abduction injury. Refer text for details.

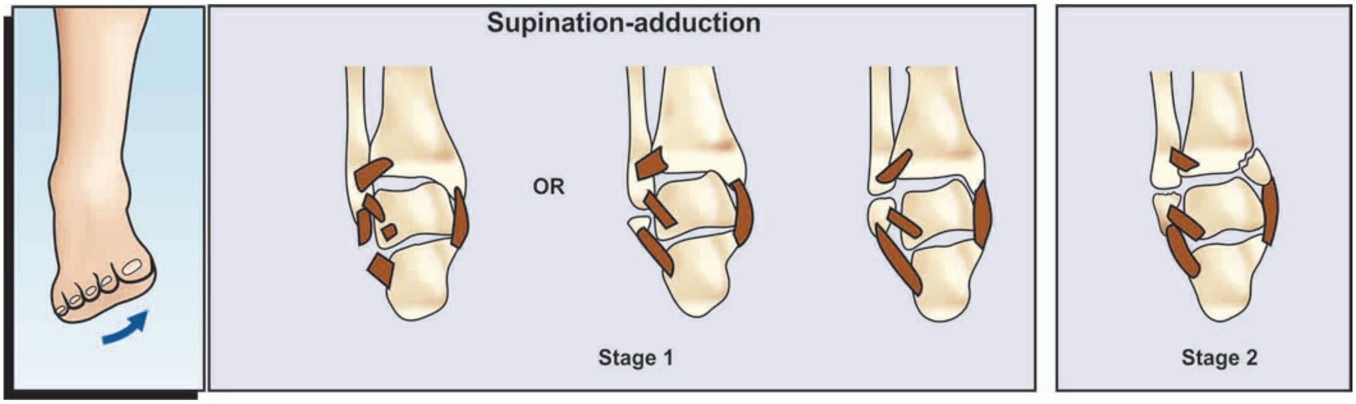


Figure 3.19
Diagrammatic representation of sequence of events occurring in a supination-adduction injury. Refer text for details.

Pronation-external rotation injuries: In these, the foot is in pronation while the talus rotates externally. This movement occurs along the long axis of the talus.

a. The medial failure pattern is the same as in abduction injuries, i.e. either there is a medial malleolar fracture or a deltoid ligament rupture. (The medial malleolar fragment is generally small when compared to that occurring in abduction injury—Stage 1)
b. The talus which is free of its medial tether, springs forwards and out of the ankle mortise. This exerts *stress on the* syndesmotic ligaments which bind the inferior tibiofibular syndesmosis. The first to fail is the anterior tibiofibular ligament, followed by interosseous tibiofibular ligament. As a result, partial diastasis of inferior tibiofibular syndesmosis occurs. If the force continues to act, the fibula starts rotating on the intact posterior tibiofibular ligament using it as a pivot. The resultant stress on the fibula, may cause an oblique/spiral fracture which can occur as low as lower 1/3rd of the fibula to as high as fibular neck. This failure pattern is known as *Maisonneuve Injury—Stages 2 and 3 (Fig. 3.20).* (Named after *Jules Germain Francois Maisonneuve* who described the mechanism)
c. At this juncture, when partial diastasis has occurred if the subject is running, e.g. in an athletic event, the talus is vertically pushed upwards between the tibia and the fibula. This ruptures the intact posterior tibiofibular ligament causing complete diastasis of inferior tibiofibular syndesmosis and a comminuted fracture of the lower 1/3rd of the fibula. This is known as *Dupuytren's fracture*. (Figs 3.21A and B)—Stage 4. (Named after *Guillaume Dupuytren* who described the mechanism in 1816)

Tillaux fracture (Named after *Paul Jules Tillaux* who described this fracture in 1848). It is a fracture occurring during the diastasis of inferior tibiofibular syndesmosis. If the strength of the tibiofibular ligament is more than that of the bone, there develops an avulsion fracture at the anterior/posterior lip of the inferior fibular facet of the tibia. Anterior lip fracture is known as Anterior *Tillaux fracture: see (Fig. 3.20) marked—Stage 2 B* and Posterior lip fracture is known as posterior *Chaput-Tillaux* fracture described by *Chaput. See (Fig. 3.20) marked—Stage 4 D*.

Complete failure pattern in pronation external rotation injury is either Maisonneuve or Dupuytren's fracture-dislocation.

Incomplete failure presents only with those injuries that exist at the time when the deforming force ceases to act. (Deltoid ligament rupture, medial malleolar fracture, diastasis of syndesmosis, etc.).

Uncommon and variable pattern can present with only rupture of ligaments or a combination of rupture of ligaments and fractures.

Supination-external rotation injuries: In these, the foot is in supination while the talus rotates externally. When the talus supinates and rotates externally, it springs backwards and out of the ankle mortise. Hence, the failure sequence that occurs is as follows (Figs 3.22 and 3.23):

- First structure to fail is the lateral malleolus—Stage 1
- Next is the posterior malleolus—Stage 2
- Last is the medial malleolus—Stage 3.

The fracture is known as a trimalleolar fracture or a 'Cotton's fracture'. The direction of the fracture line in these fractures is obliquely upwards and backwards.

Complete failure pattern is a trimalleolar fracture or a 'Cotton's fracture' described by Cotton FJ in the year

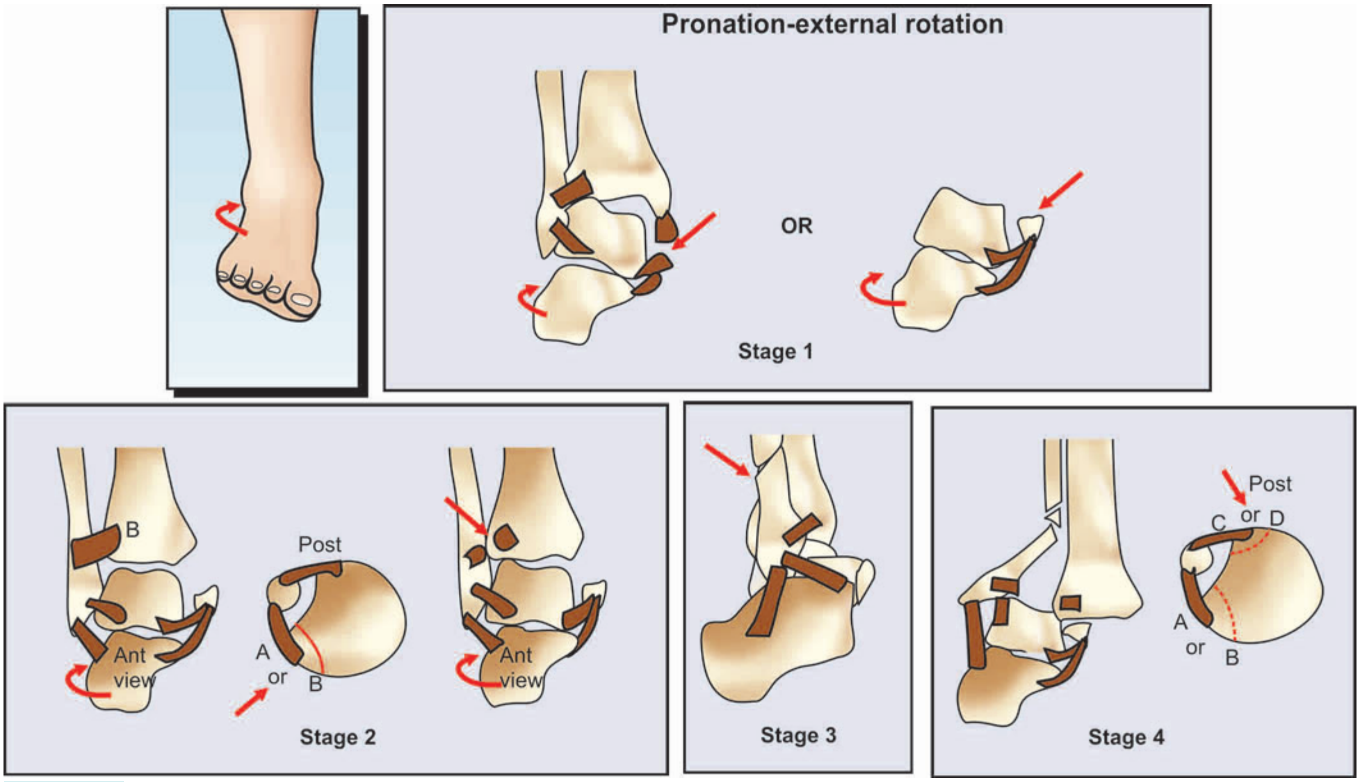
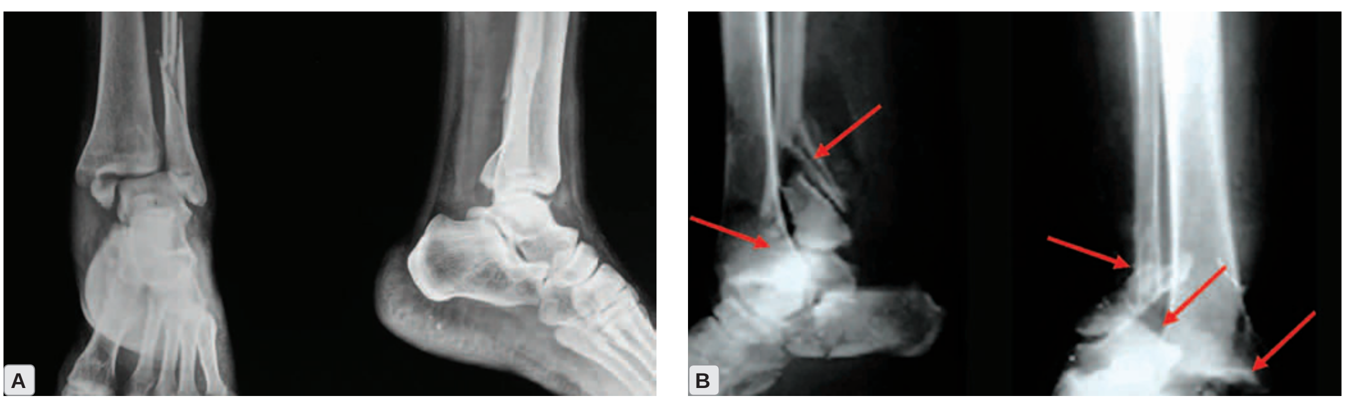


Figure 3.20

Diagrammatic representation of the sequence of events occurring in a pronation-external rotation injury. Stage 1 indicates rupture of deltoid ligament or medial malleolar fracture. Stage 2 indicates rupture of anterior tibiofibular ligament (marked A) OR anterior Tillaux fracture (marked B). Similarly in Stage 4 indicates posterior tibiofibular ligament rupture (marked C) OR posterior Chaput-Tillaux fracture (marked D). Refer text for details.



Figures 3.21A and B

(A) AP and lateral view of the ankle showing a closed Dupuytren's fracture dislocation; (B) AP and lateral views showing an open Dupuytren's fracture dislocation. Presence of foreign bodies in the X-rays indicates the compound nature of the injury.

1915. Incomplete/variable failure pattern may manifest with isolated fractures of the malleoli or any two of them together.

Direction of the fracture line as well as posterior displacement of the talus, gives the clue about the mechanism of injury.

Vertical compression injuries: Occurs when a person falls from a height. The talus is violently pushed upwards into the inferior articular surface of the tibia, shattering the articular surface. Extent of disruption is directly proportional to the severity of the impact. The term Pilon fracture is given for these injuries (Fig. 3.24).

Figures 3.22A to C

(A and B) Cotton's fracture; and (C) Radiograph of the same treated by ORIF using cannulated cancellous screws for the medial malleolus and cortical screws for the lateral malleolus (Interfragmentary screw fixation). The posterior malleolar fragment is less than 1/3 of the articular surface and has fallen back into place with reduction and did not require fixation. Note the backward movement of the talus in radiograph A which was responsible for fracture of the third malleolus. This backward displacement also indicates that the mechanism of injury is supination external rotation.

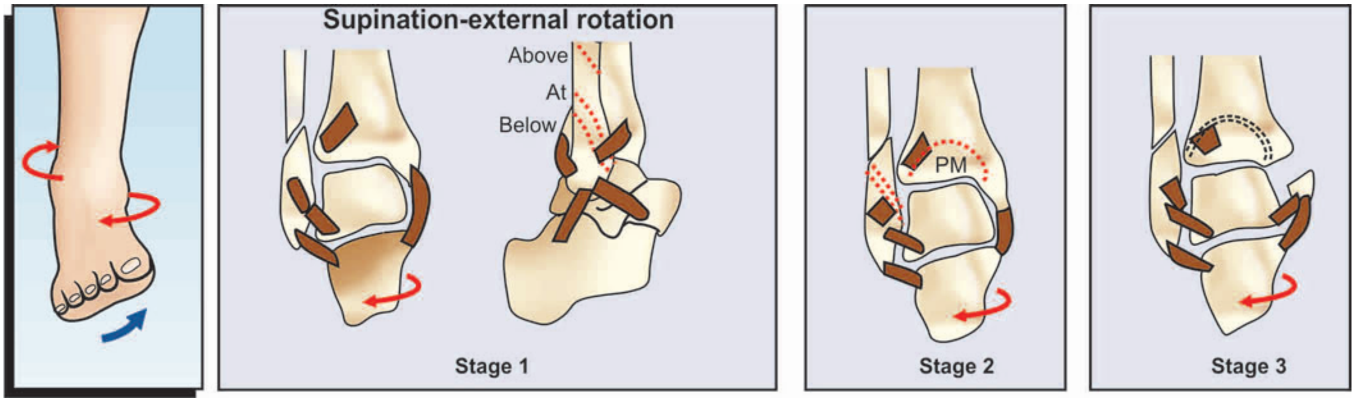


Figure 3.23

Diagrammatic representation of sequence of events occurring in supination-external rotation injury. Refer text for details. *Posterior malleolus—PM, shown in red dotted lines.

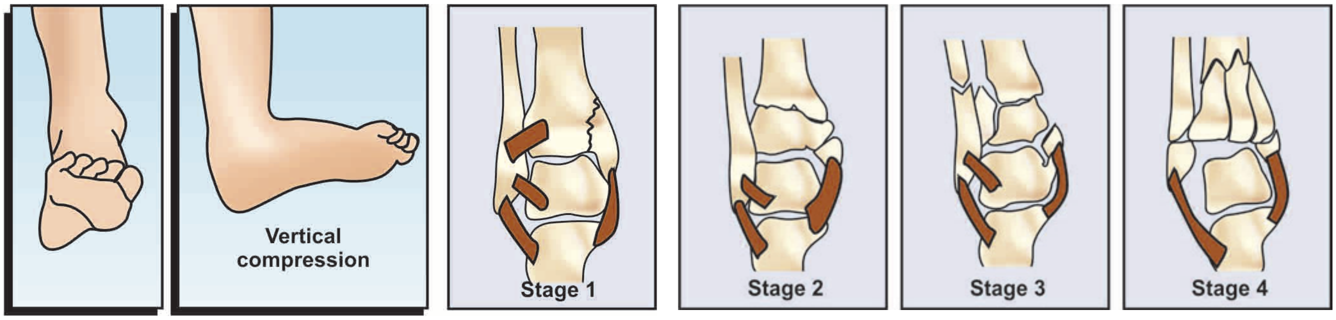


Figure 3.24

Diagrammatic representation of the stages of a vertical compression injury. Refer text for details.

Figure 3.25

An ankle joint involved in a high velocity injury of a road traffic accident. The injury is a compound fracture dislocation. Mechanism is simply unclassifiable.

Unclassifiable injuries: Nature of injury is so severe that the mechanism is unclassifiable. This is because a variety of forces acting simultaneously or in succession have caused the failure (Fig. 3.25).

These injuries are always compound/open injuries resulting in severe morbidity.

Management (Figs 3.26A to C)

Nonoperative

Undisplaced and incomplete fractures: They are managed with immobilization in plaster/fiber, below knee or above knee cast as per indication for a period of 4–6 weeks.

Operative

Displaced fractures: They are managed by open reduction and internal fixation with emphasis on restoration of articular surface congruity.

Figures 3.26A to C

Different methods of fixation of ankle fractures: (A) An isolated fracture of the medial malleolus fixed internally using a malleolar screw; (B) A malleolar fracture with a small malleolar fragment. This was fixed using tension band wiring; (C) A vertical compression injury with Pilon fracture internally fixed using malleolar screws, cancellous screws and cortical screws. The fibular fracture was fixed using 1/3rd tubular plate and screws. The inferior tibio-fibular syndesmosis was transfixed.

The Surgical Sequence of Reconstruction Followed in Vertical Compression Injury

i. Fixation of fibular fracture.
ii. Reconstruction of tibial plafond articular fracture.
iii. Autologous bone grafting if necessary.
iv. Anteromedial tibial plate fixation.

Mobilization of the joint is to be started at the earliest after wound healing.

Implants used: Malleolar screws, cannulated cancellous screws, 1/3rd tubular plates, 3.5 mm LC DCP, Rush nails, K-wires and SS wires (for tension band wiring).

Biodegradable screw is the latest among implants.

Complications

- Nonunion—seen when the injury is neglected.
- Improper restoration of articular congruity by neglect or otherwise (e.g. Pilon fracture) results in painful limitation of movement and degenerative arthritis at a later date.

Treatment

- Nonunion is treated by open reduction internal fixation and bone grafting.
- Degenerative arthritis is treated by arthrodesis of the ankle joint or total joint replacement.

Revision Questions

Q. Define Pott's fracture.
Q. What is Maisonneuve injury?
Q. What is Dupuytren's fracture?
Q. What is Cotton's fracture?
Q. What is Tillaux fracture?
Q. What are various implants used to fix the fracture dislocation of the ankle?

Essay Question

Q. Discuss the classification, mechanism of injury, diagnosis and management of fracture dislocation of ankle otherwise called as Pott's fracture. Enumerate its complications.

TIBIAL DIAPHYSEAL FRACTURES

Almost 1/3rd of the tibia is subcutaneous, i.e. its anteromedial border (known as shin) and the flat medial surface. Hence, this bone is predisposed for open fractures. The nutrient artery which arises from posterior tibial artery enters the bone just distal to the origin of soleus muscle and runs distally (from the knee I flee.) Hence, chances of delayed union and nonunion increases especially in more distal fractures because of poor blood supply.

Mechanisms of Injury

Direct: This mechanism operates in a high energy trauma, e.g. road traffic accidents, and penetrating injuries. Open, comminuted, displaced fractures and crush injuries (injury to the bone and soft tissue) are commonly seen.

Indirect: This mechanism operates in a low energy trauma, e.g. slip and fall. The resultant torsional stress causes oblique, spiral and minimally displaced fractures, with minimal soft tissue damage.

The injury is considered as more severe when the fracture pattern is comminuted or spiral than when it is transverse.

Management

Methods employed are *nonoperative and operative.*

Nonoperative: Less severe forms of fractures are treated with closed reduction and maintenance of reduction in an above knee (AK) plaster of Paris or fiber cast. PTB cast/the Sarmiento technique of cast bracing is employed to enhance healing when delayed union is observed at follow-up.

Sarmiento technique (Fig. 3.27): The technique is based on the principle of encouraging micromovement at the fracture site to stimulate the process of healing by giving a patellar tendon bearing (PTB) cast. This cast is given when early union is seen after nonoperative treatment of tibial diaphyseal fracture with an AK cast.

The PTB cast is moulded all round the patellar tendon to become effective. Also, a walking rocker sole is attached to the foot end. Partial weight bearing is encouraged as tolerated after giving the PTB cast. The fracture is then evaluated after 3–4 weeks for progression of union.

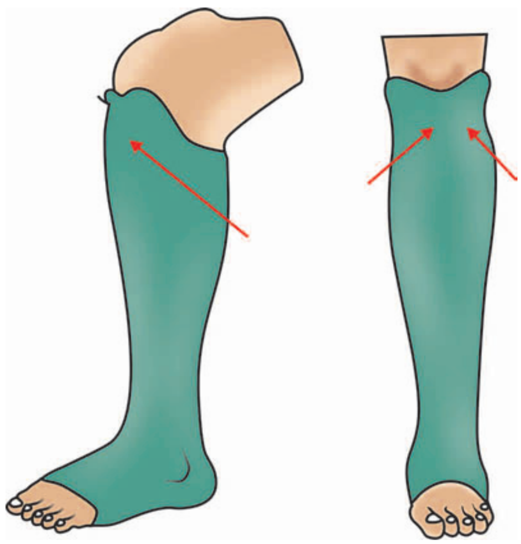
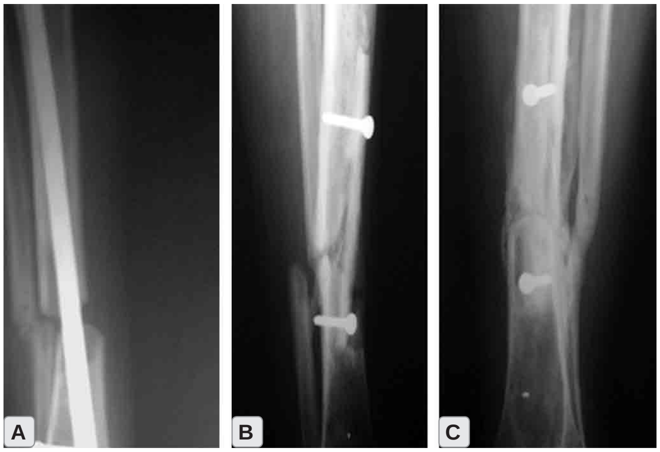


Figure 3.27

Diagram of a Patellar tendon bearing (PTB) cast. Note the moulding at the patellar tendon for weight transmission during walking. The popliteal fossa should always be left free of plaster.



Figures 3.28A to C

(A) Type III open fracture with bone loss. Initially stabilized by an intramedullary interlocking nail to save the limb. Remained ununited at the end of three months: by then soft tissue cover had been obtained and infection controlled; (B and C) Successful treatment by corticocancellous grafting using sliding cortical graft from the same tibia to bridge the defect and cancellous grafts from the iliac crest to induce osteogenesis. Note the sound union with adequate new bone formation at the end of 6 months (3 months following bone grafting).

Operative:

1. Closed/open reduction and internal fixation is the procedure of choice in more severe, displaced, comminuted and segmental fractures. Generally, ender nails and interlocking nails are the implants that are used.
2. Plates are also used by employing the technique of MIPPO (Minimally Invasive Percutaneous Plate Osteosynthesis).
3. External fixator application after thorough wound debridement is indicated in open fractures to save the limb, facilitate good postoperative wound care and undertake secondary flap coverage procedure.

Complications and their Management

Common complications are:

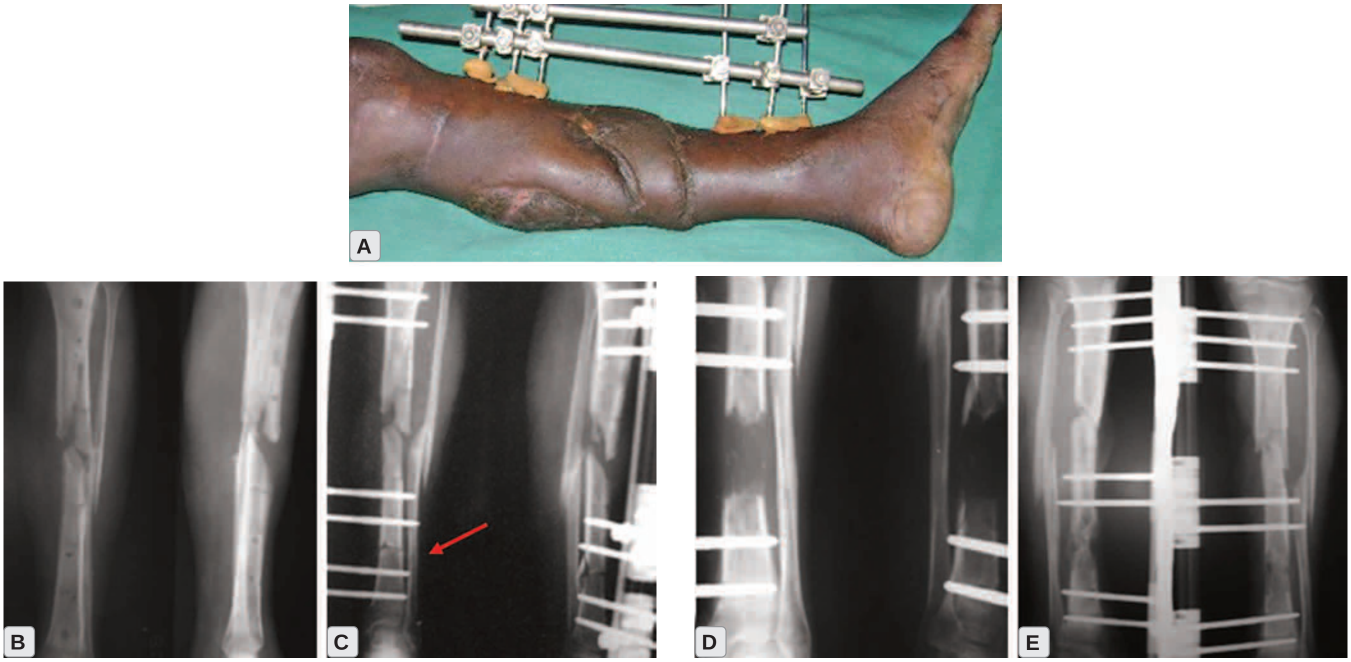
a. Compartment syndrome
b. Delayed union
c. Nonunion

Compartment syndrome is managed by immediate adequate decompression of involved leg compartments along with appropriate skeletal stabilization as per indication (External/internal).

Delayed unions and nonunions are treated by bone grafting techniques (cancellous/corticocancellous grafting—Refer chapter 1) along with stabilization by means of internal fixation or immobilization in plaster/fiber cast (Figs 3.28A to C).

In difficult cases with bone loss and bad scarring, corticotomy and bone transportation (Refer chapter 1) is employed using Ilizarov, Rail fixator or any other appropriate fixator (Figs 3.29A to E).

Note: ***Open tibial fractures should always be converted into closed fractures as early as possible in order to prevent: (a) leak of fracture hematoma, (b) devitalization of exposed bone and (c) secondary infection; thereby facilitating normal healing process and preventing the complication of delayed union, nonunion and osteomyelitis. Many times, it is not possible to achieve an early soft tissue cover and nonunion OR delayed unions are common. These complications are expected and accepted in open tibial diaphyseal fractures and are treated accordingly.***



Figures 3.29A to E

(A) Result after sural flap cover for a Grade III open fracture stabilized primarily by external fixator; (B) Persistent defect secondary to bone loss; (C to E) Distraction and compression osteogenesis by bone transportation technique using uniplanar fixator following a lower tibial corticotomy. Because of bad scarring over the anteromedial and lateral aspect of the tibia it was decided to employ this technique. (E) Note the successful result.

> *Essay Question*
>
> Q. Classify open fractures. Discuss the management of an open diaphyseal fracture of the tibia and its complications.

INJURIES AROUND THE KNEE JOINT

Injuries around the knee joint include:

a. Ligament injuries
b. Meniscal injuries
c. Fractures

Ligament Injuries

The principal ligaments which stabilize the knee joint are the medial and lateral collateral ligaments as well as the anterior and posterior cruciate ligaments.

Anatomy and Pathomechanics of Ligament Disruptions

a. *Medial collateral ligament:* Extends from medial femoral condyle to medial tibial condyle. Has two components: (i) superficial and (ii) deep. The deep component is divided further into the meniscofemoral and meniscotibial ligaments by its attachment to medial meniscus. It is the main stabilizer of the knee in 30° of flexion. It resists valgus stress. Hence, an excessive valgus stress tears or ruptures the ligament (Fig. 3.30).
   Thus, the test employed to diagnose the ruptured medial collateral ligament is known as 'Valgus stress test' (Fig. 3.31).
b. *Lateral collateral ligament:* Extends from lateral femoral condyle to the head of the fibula. It is not attached to the lateral meniscus and is separated from it by the tendon of the popliteus muscle.
   It resists varus stress. Hence, an excessive varus stress tears or ruptures the ligament.
   Thus, the test employed to diagnose the ruptured lateral collateral ligament is known as 'Varus stress test' (Fig. 3.32).
c. *Anterior cruciate ligament:* Extends from the anterior intercondylar eminence of the tibial plateau to the inner aspect of the lateral femoral condyle. Because the ligament twists on itself before getting attached to the femur, its anterior fibers are posterior at insertion and posterior fibers are anterior. It is thus

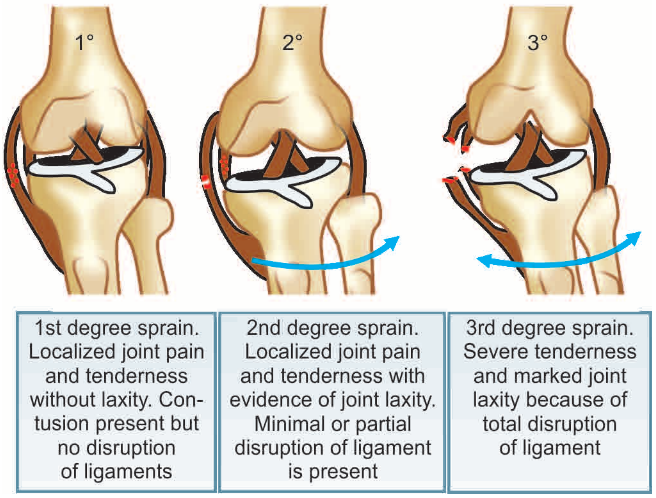


Figure 3.30
Different types of medial collateral of ligament disruption that can occur following a valgus stress.

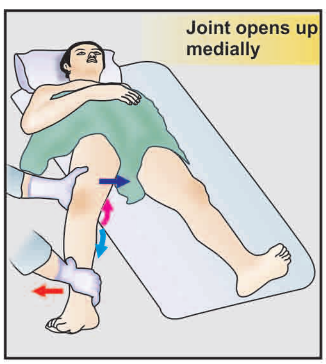


Figure 3.31
Performance of a valgus stress test.

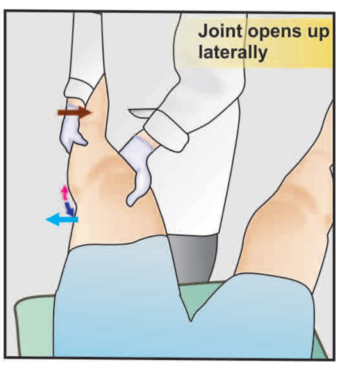


Figure 3.32
Performance of a varus stress test.

divided into two components, the anteromedial and the bulk. The anteromedial portion becomes taut in flexion and the bulk portion becomes taut in extension thus imparting anterior translational stability during flexion as well as during extension of the knee joint. It also prevents the femur from sliding posteriorly on the tibia, prevents hyperextension of the knee and limits medial rotation when the foot is on the ground. It gets injured when subjected to a twisting force on a hyperextended knee (Figs 3.33A and B).

The tests employed to diagnose anterior cruciate ligament injury are the 'Anterior drawer test' and the 'Lachman test.' They demonstrate anterior translation of the tibia on the femur. 'Lachman test' is more sensitive. Meniscus may sometimes block the anterior translation of the tibia in anterior drawer test (Figs 3.34 and 3.35) resulting in a false negative test.

d. *Posterior cruciate ligament:* Extends from the posterior intercondylar eminence to the inner aspect of the medial femoral condyle. It imparts posterior translational stability during flexion and extension of the knee. It also prevents the femur from sliding anteriorly on the tibia and limits lateral rotation when the foot is on the ground. It gets injured when there is a posterior thrust on a hyperextended knee, e.g. stepping into a pot hole or a direct blow onto the upper tibia (Fig 3.36).

The test employed to diagnose the posterior cruciate ligament injury is the 'Posterior drawer test'. It demonstrates posterior translation of the tibia on the femur (Fig. 3.37).

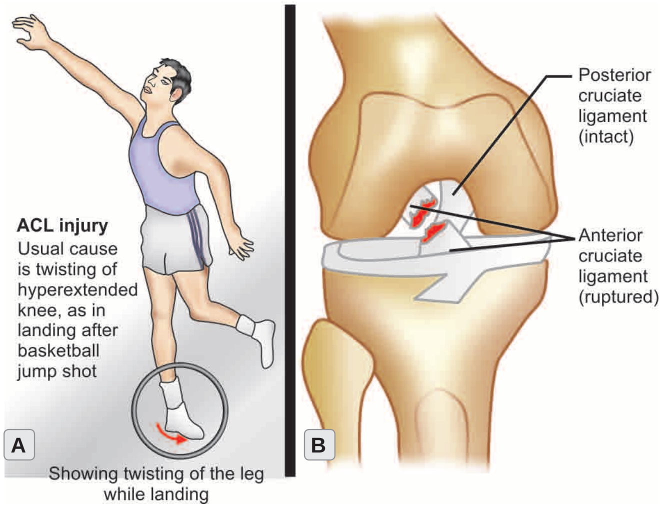
Terrible Triad of O'Donoghue (Unhappy Triad)

It is a combination of injuries involving the medial meniscus, medial collateral and anterior cruciate ligaments which results in a highly unstable knee. It needs highly skilled, professional management (Fig. 3.38).

Management of Ligament Injuries

a. *Primary repair:* Repair is done as an immediate procedure within 24–48 hours after the injury. After repair, the limb is immobilized in a cast for a period of 3–6 weeks followed by gradual and active mobilization and muscle strengthening exercises.
b. *Secondary reconstruction*: It is done after 3–6 weeks OR when the patient presents late after injury, as a secondary procedure. Tendon and fascial grafts are used to substitute the torn ligaments, e.g. semitendinosus, bone patella bone graft, fascia lata. Sometimes, when donor tendons are not available, artificial ligaments are used. The limb is immobilized in a knee brace and is mobilized gradually after 48 hours.

Both the above procedures can be done by means of an open or arthroscopic assisted technique, depending on the indication and proficiency of the operating surgeon.



Figures 3.33A and B

Mechanism of an anterior cruciate ligament (ACL) injury.

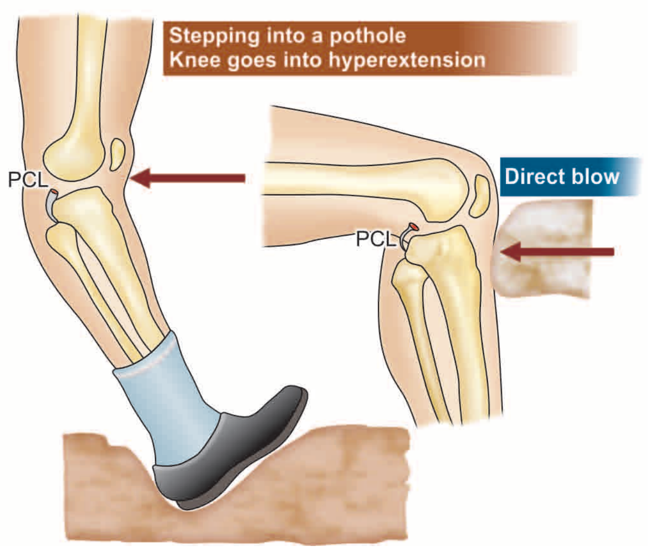


Figure 3.36

Mechanism of posterior cruciate ligament (PCL) injury. Refer text for details.

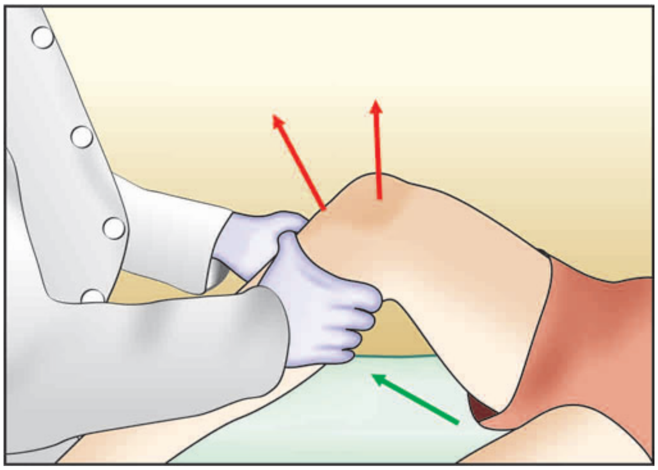


Figure 3.34

Anterior drawer test. Refer text for details.

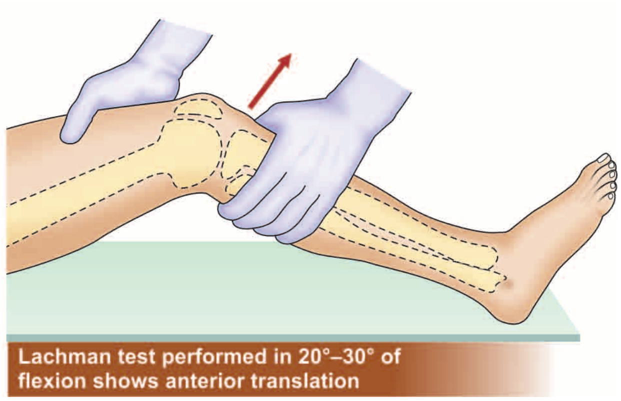


Figure 3.35

Lachman test. Refer text for details.

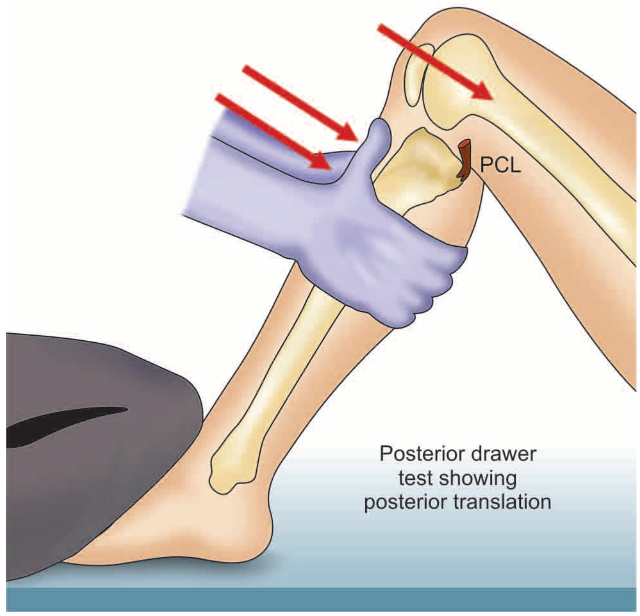


Figure 3.37

Posterior drawer test. Refer text for details. *Posterior cruciate ligament—PCL.

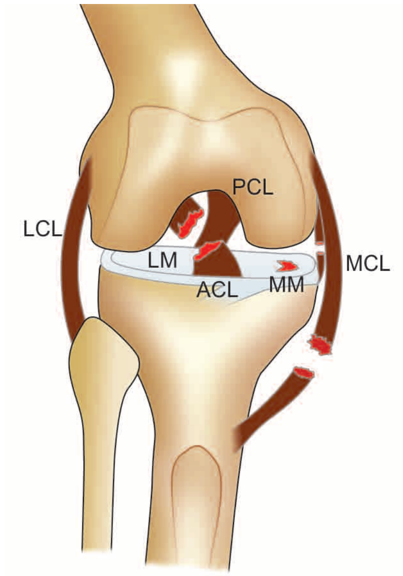


Figure 3.38

'Terrible/unhappy triad' of O'Donoghue. In this medial collateral ligament, anterior cruciate ligament and medial meniscus are damaged. *Medial collateral ligament—MCL; Lateral collateral ligament—LCL; Anterior cruciate ligament—ACL; Posterior cruciate ligament—PCL; Medial meniscus—MM; Lateral meniscus—LM.

Revision Questions

Q. Valgus stress test.

Q. Varus stress test.

Q. Anterior drawer test.

Q. Posterior drawer test.

Q. Lachman test.

Essay Question

Q. Discuss ligament injuries of the knee joint and their management.

Meniscal Injuries

'Meniskos' in Greek means Crescent. The meniscus is a fibrocartilaginous semilunar (C-shaped) structure situated within the knee joint which is moulded according to the shape of the tibial and the femoral condyle, being convex on its undersurface and concave on its surface, thicker at the periphery and thinner at the center. It is attached to the tibia along its anterior (anterior horn), lateral and posterior (posterior horn) margins but is free in its medial margin. The blood supply to the meniscus is more towards the periphery than at the center (Hence, peripheral tears heal better when compared to central tears).

Functions of the Meniscus

During the movements of the knee and weight bearing, the meniscus:

1. Prevents friction by cushioning effect.
2. Helps in the distribution of synovial fluid.
3. Helps in the distribution of body weight.

Loss/removal of the meniscus results in impairment of above functions and predisposes to degenerative arthritis.

An injured meniscus too, leads to derangement in the congruity of the joint, interferes with normal movements and promotes uneven distribution of stresses, thus accelerating degenerative changes.

Medial meniscus is larger in diameter, has a narrow body and is less mobile because it is attached to the deep portion of the medial collateral ligament through the meniscofemoral and meniscotibial ligaments.

Lateral meniscus is smaller in diameter, has a wider body and is more mobile because it is not attached to the lateral collateral ligament but separated from it by the tendon of popliteus.

For these reasons, medial meniscus is injured more commonly than the lateral meniscus (Figs 3.39 to 3.41).

Mechanism of Injury

During normal flexion and extension of the knee joint, the menisci follow the femoral and the tibial condyle and allow a smooth gliding. But when there is an abnormal stress of rotation, instead of following the condyles they start moving on the condyles. Thus, they are drawn into the center of the joint where they get trapped, pinched and torn. Flexion, abduction, external rotation stress causes internal rotation of femur on the tibia and draws the medial meniscus—to the center and injures it. Flexion, adduction, internal rotation stress causes external rotation of femur on the tibia which draws the lateral meniscus to the center of the joint and injures it. The site of tear is related to the degree of flexion. In greater degree of flexion, posterior part of the meniscus (posterior horn) is involved. In lesser degree of flexion, anterior part of the meniscus is involved. Thus, the tear begins posteriorly and extends from posterior to anterior direction as the knee extends from a flexed position.

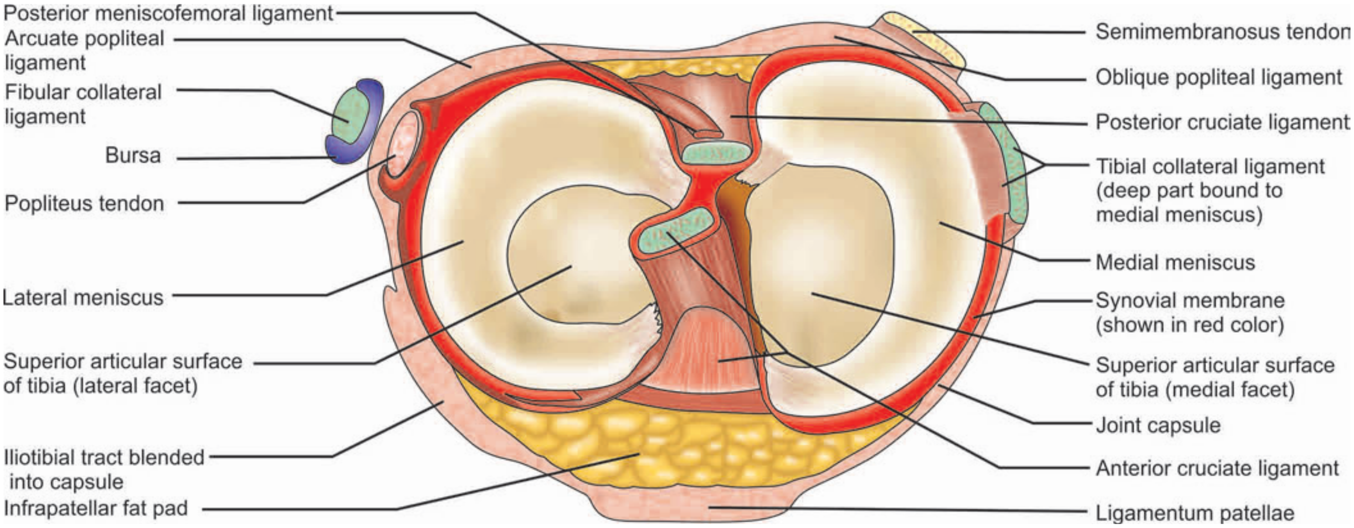


Figure 3.39

Diagrammatic representation of the structures on the tibial plateau.

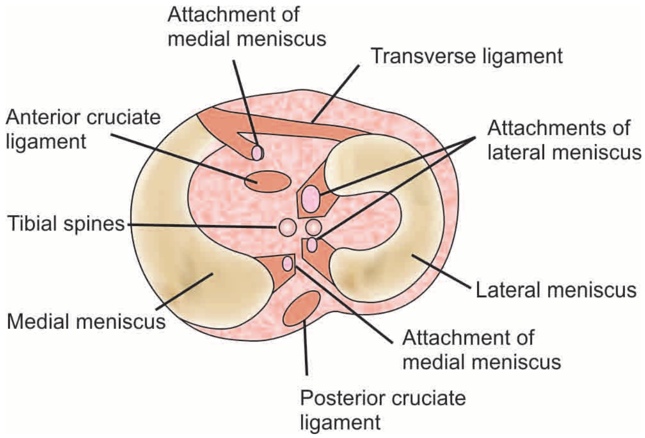


Figure 3.40

Structures attached to the upper surface of the tibia.

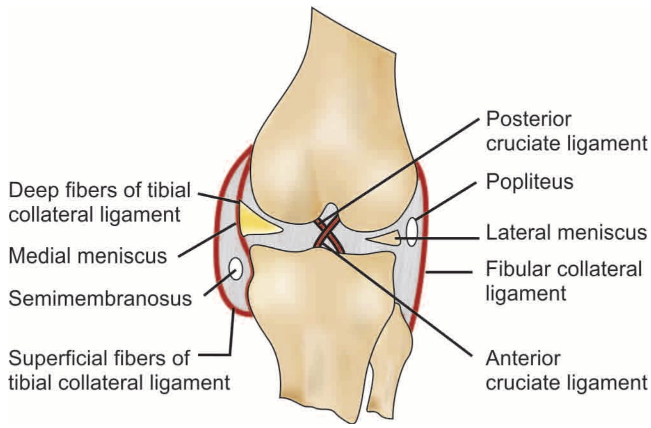


Figure 3.41

Diagrammatic representation of normal anatomy of the knee.

Types of tear (Fig. 3.42)

A. Longitudinal tear—May proceed to become a ‘Bucket handle tear’.
B. Radial tear—May proceed to become a ‘Parrot beak tear’.
C. Horizontal tear—May proceed to become a ‘Flap tear’.
D. Combined tear—A combination of different tears, generally seen in a degenerative meniscus (in elderly).

Diagnosis

Signs and symptoms

a. Constant pain in the knee, aggravated by movement.
b. Tenderness in the joint line (Medial/Lateral).
c. Swelling when effusion is present.
d. Sensation of giving way.

> Other causes for sensation of giving way are:
> 1. Loose bodies.
> 2. Chondromalacia patella.
> 3. Weakness of quadriceps muscle.
> 4. Instability due to ligament injury.

e. Pathological locking. This is seen only in ‘Bucket handle tear’. Pseudolocking is seen in acute injuries of the knee with hemarthrosis.

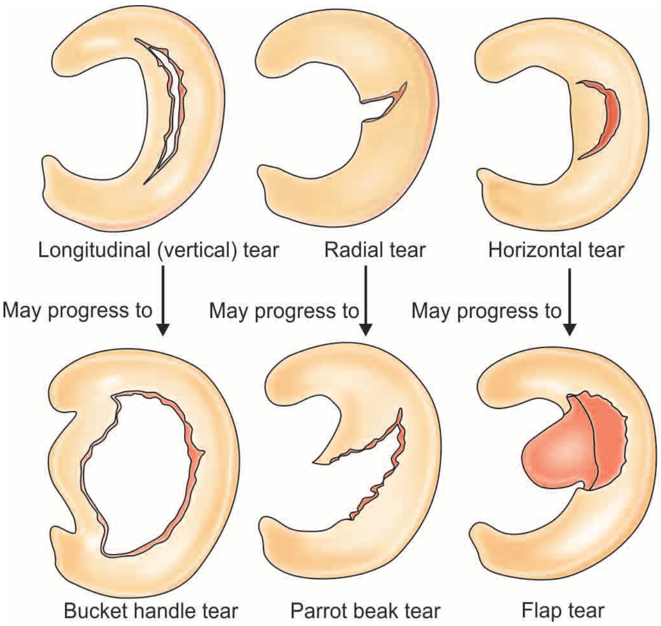


Figure 3.42

Different types of meniscal tear.

Note: *Sensation of giving way and pathological locking occur when the torn portion of the meniscus comes in contact with the articular surface of the femur during movement. In pathological locking, knee remains fixed in a particular degree of flexion. Any attempted movement is painful (except passive extension which is possible at times). It becomes free only when it gets unlocked by itself or by maneuvers. Sensation of giving way, is a feeling of instability below the knee, as if weight transmission has ceased below the knee and the limb below the knee is disconnected from the proximal part.*

Diagnostic Clinical Tests

1. *McMurray's test*
   The test aims at reproducing the mechanism of injury by gentle maneuvering.
   a. *For medial meniscus:* With the patient lying supine on the examination table, the *knee is completely flexed.* To check the medial meniscus, the examiner *palpates the posteromedial joint with one hand and holds the foot with the other. The leg is then externally rotated and the knee is gradually extended giving a valgus stress. A click is appreciated* when the torn portion of the medial meniscus comes in contact with the femur.
   b. *For lateral meniscus:* With the patient lying supine on the examination table, the *knee is completely flexed.* To check the lateral meniscus, the examiner *palpates the posterolateral joint with one hand and holds the foot with the other. The leg is then internally rotated and the knee is gradually extended giving a varus stress. A click is appreciated* when the torn portion of the lateral meniscus comes in contact with the femur.

   Note:
   i. *The tear of the posterior portion of the meniscus produces a click during initial 0–90°of movement, as the knee extends from a completely flexed position. The tear involving the middle and anterior portion of the meniscus produces a click beyond 90° of movement i.e. from 90° to full extension.*
   ii. *The medial meniscus is more commonly injured than the lateral meniscus. The click appreciated is not only felt but can be audible at times.*
2. *Apley's grinding test*: With the patient prone, the knee is flexed to 90° and the thigh is fixed against the examination table. The leg is then held, pulled upwards and a rotational strain is given. When ligaments are torn, this part of the test is painful. Next, the leg is pushed downwards and rotated while the joint is slowly flexed and extended. When the meniscus is injured, pain is observed in the joint during this maneuver. Thus, the test helps in differentiating ligament injuries and meniscal injuries.
3. *Squat test*: This test is performed by asking the patient to take full squats with the leg and the feet together, alternately keeping the lower limb in internal and external rotation. Pain in the joint in internally rotated position (external rotation of femur on the tibia) suggests injury to the lateral meniscus and pain in externally rotated position (internal rotation of femur on the tibia) suggests injury to the medial meniscus.

Note: *Presence of pain and click are diagnostic of meniscal injury. But absence doesn't rule out tears of the meniscus.*

Investigations

a. *Plain X-ray:* Is done mainly to identify associated fractures. It is useful in long-standing injuries to look for degenerative changes in the knee joint.
b. *Diagnostic arthroscopy:* Is an ideal and confirmatory investigation. By arthroscopy, other associated

injuries, e.g. osteochondral fractures can also be identified. (If necessary, to be followed immediately with arthroscopic surgery in the same sitting as this is an invasive investigation and is done under anesthesia)

c. *MRI:* Is a noninvasive procedure. It needs a good sensitive machine as well as accurate interpretation to avoid false negative and false positive reports.
d. *Arthrography:* After the advent of arthroscopy, arthrography has a very limited role and is not done routinely.

Management

Absence of meniscus does not compromise the function of the knee markedly. It only leads to late degenerative changes. But an injured/torn meniscus severely compromises the function of the knee. As this is directly proportional to the severity of injury, it needs immediate attention.

When possible, the torn meniscus is repaired, e.g. smaller tears. The procedure is known as *meniscorraphy.*

When repair is not possible, the torn meniscus is excised either partially, e.g. in larger tears involving posterior horn, bucket handle tear, etc. OR completely, e.g. in tears with a degenerate meniscus, complex tear, etc. The procedure is known as *meniscectomy (partial/ complete.).* It is usually done by arthroscopic surgery. It can also be done by open surgical methods.

Note: *Complete/total meniscectomy is rarely done these days. It is only the damaged portion of the meniscus that is removed (partial meniscectomy). Peripheral attachment is always preserved.*

Current research is towards meniscal replacement using meniscus obtained either from a brain dead cadaver or using an artificial meniscus.

Revision Questions

Q. McMurray's test.
Q. Apley's grinding test.
Q. Discuss the mechanism of meniscal injury.

Essay Questions

Q. Discuss the anatomy of the knee joint. Classify the meniscal injury and discuss its management.
Q. Discuss meniscal injury with reference to the mechanism, classification, diagnosis and management.

Fracture of the Patella

General Information

Patella is a sesamoid bone within the quadriceps muscle. It was thought to be a phylogenetically inherited bone without any function (Brooke, 1937). This theory, resulted in removal of the fractured patella and patellectomy became the treatment of choice until Haxton (1945) proved beyond doubt that the patella has a definite function. The patella acts as a pulley, increases the moment arm of the extensor mechanism (the distance from the muscle to the mechanical axis of the knee) and augments the power of the quadriceps by increasing it. This is the main function of the patella. After patellectomy, much of this power is lost and quadriceps becomes weak resulting in a quadriceps lag (Quadriceps stops short of terminal 5° of extension). Other functions of the patella are

i. It gives protection to the knee.
ii. It helps in cosmetics of the knee.
iii. It helps in centralizing the forces coming from four units of quadriceps muscle.

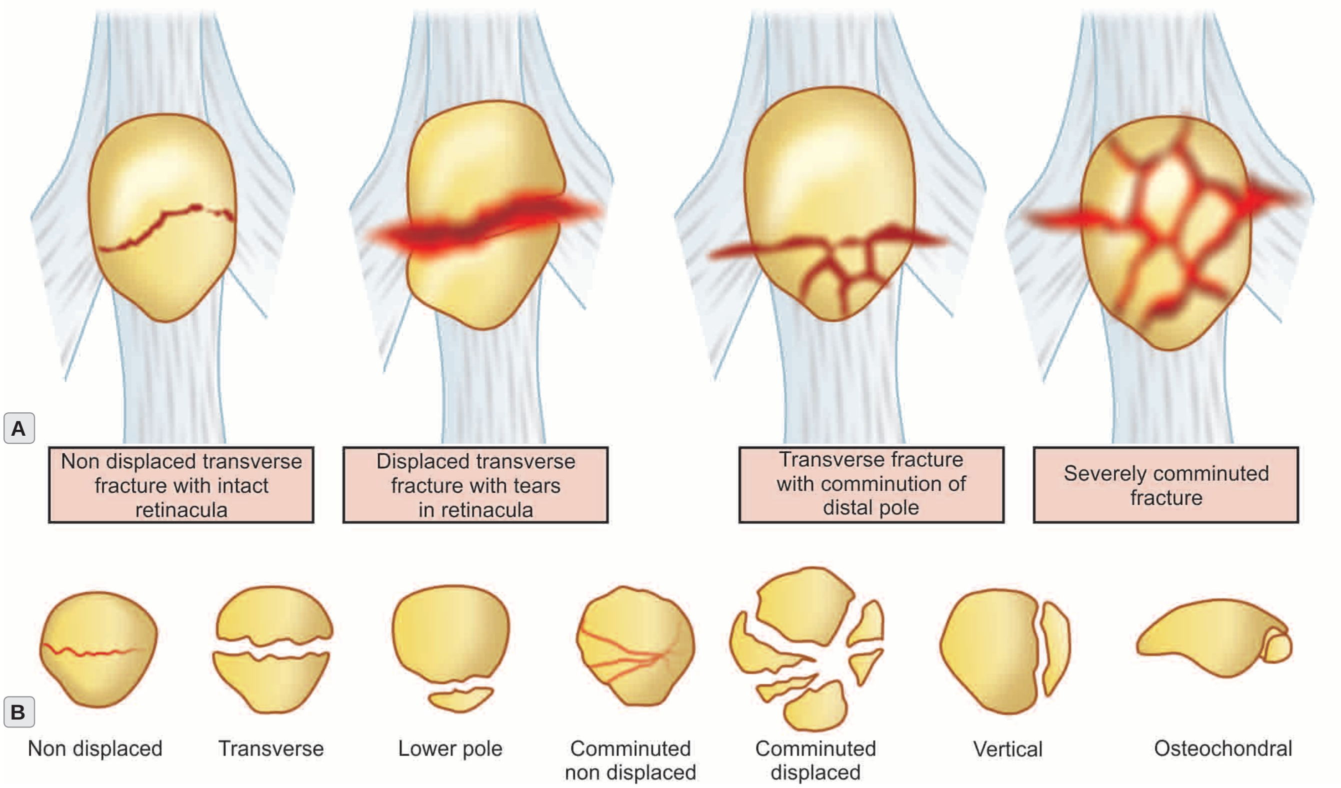
Mechanisms of Injury (Figs 3.43A and B)

a. *Direct:* In this, there is a *direct impact on the patella,* e.g. dash board injury, direct blow during an assault. The type of fracture seen, is a comminuted (shattered into pieces) fracture. The classical variety is known as a '*Stellate fracture'.*
b. *Indirect*: In this, the force acts indirectly, e.g. slipping while walking. When a person slips, one limb is off the ground and the limb which is on the ground, goes into progressive flexion at the knee. If this flexion continues it results in a fall. A reflex mechanism operates to avoid fall wherein the flexed limb tries to straighten out in order to maintain balance. This straightening occurs against gravity and the body weight. Thus patella breaks. The type of fracture that occurs is transverse in nature.
c. *Combined mechanism:* The major fragment remains non-comminuted and the other fragment is comminuted (i.e. the minor fragment).
d. *Avulsion:* It occurs due to the pull of quadriceps muscle or patellar tendon and results in an isolated fracture of the superior or the inferior pole.

Note: *Whenever a comminution is observed it suggests that a direct mechanism has operated in causing a fracture and vice versa.*

Clinical Signs and Symptoms

1. All the classical signs of a fracture are present.
2. Presence of hemarthrosis, resulting in acute swelling.
3. Broken patellar fragments can be felt by the palpating hand.



Figures 3.43A and B

Different types of fracture patella.

4. Hand can be insinuated between the fragments and palpation of femoral condyle is possible.
5. Loss of active extension at the knee (suggesting injury to the quadriceps mechanism).

Investigation

X-ray of the knee joint, standard anteroposterior (AP) and lateral views give accurate diagnosis.

Management

Patellectomy: This procedure is of historical importance and is rarely *done these days.* Only indication—perhaps is in elderly patients who have an osteoporotic bone, severe degenerative changes in the joint and badly comminuted fracture. *Never done in the young.*

Disadvatages of patellectomy

a. Normal protection to the knee is lost.
b. Quadriceps lag develops.
c. Permanent atrophy of the quadriceps.
d. Prolonged immobilization.
e. Pathologic ossification (Fig. 3.44).

Open reduction and internal fixation:
This procedure is the treatment of choice. Methods used consist of (Figs 3.45A and B)

1. Circlage wiring of Martin.
2. 'Figure of 8' tension band wiring.
3. Interfragmentary screw fixation combined with one of the above techniques (Figs 3.46 and 3.47).

Advantages of internal fixation

a. Normal protection to the knee is preserved.
b. No quadriceps lag.
c. No permanent atrophy of the quadriceps.
d. No prolonged immobilization.
e. No pathologic ossification.

Disadvatages of internal fixation

a. Possibility of a late patellofemoral osteoarthritis (rare).
b. Implant loosening and breakage.

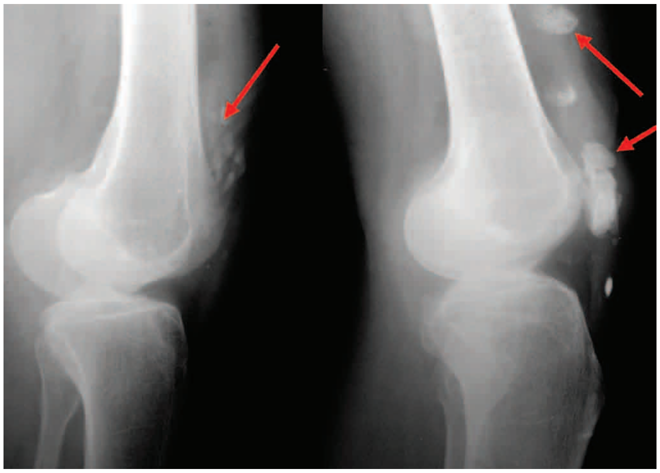
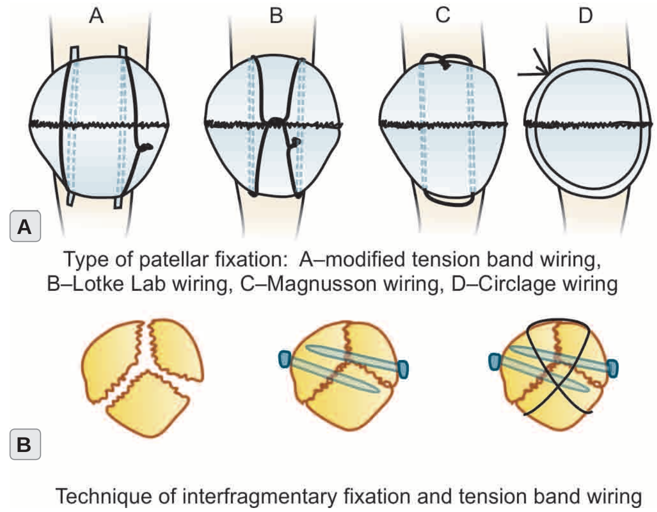


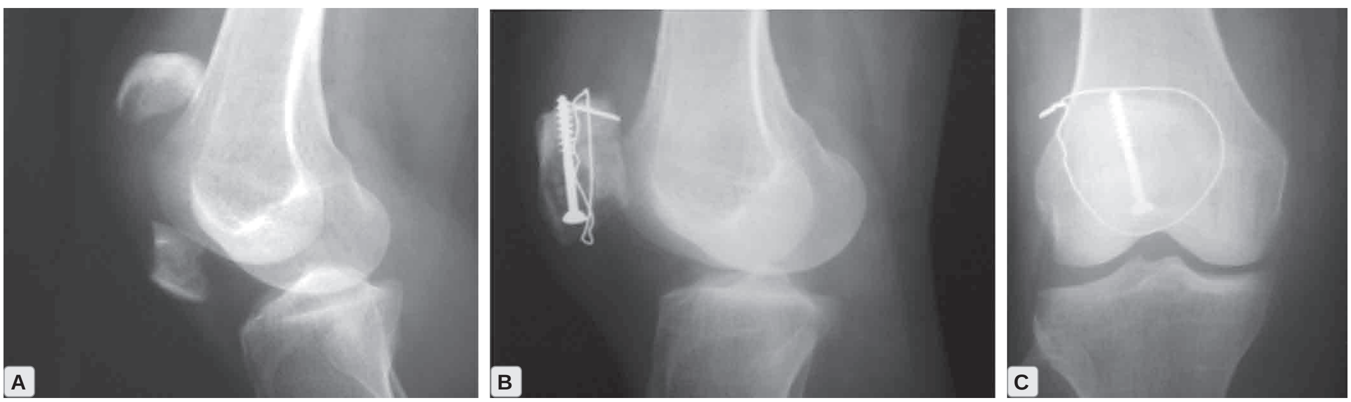
Figure 3.44

Pathologic ossification, a complication of patellectomy.



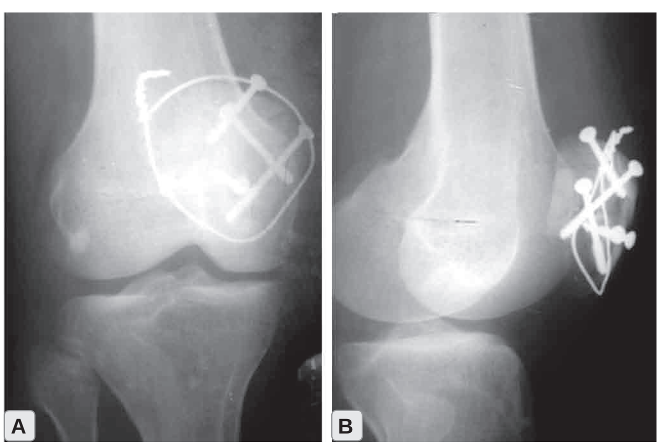
Figures 3.45A and B

Techniques employed in the fixation of fracture patella.



Figures 3.46A to C

A transverse fracture patella, fixed by a single cancellous screw and SS circlage wiring. Note the displacement of the fractured proximal fragment because of the pull of the quadriceps muscle.



Figures 3.47A and B

AP and lateral view showing a stellate fracture patella internally fixed with the help of multiple interfragmentary screws and circlage wiring.

Revision Questions

Q. Stellate fracture.

Q. Patellectomy.

Essay Question

Q. Discuss the functions of the patella. Describe the mechanism of injury in fracture patella and discuss its management.

Fractures of the Tibial Condyle (Tibial Plateau)

General Information
Tibial plateau fractures commonly occur as a result of axial loading. They are known as bumper or fender fractures because they occur as a result of an impact of the bumper on the knee, in a road traffic accident. They are often associated with ligamentous disruption of the knee. Also, injury to the nerves and vessels and compartment syndrome is quite common with these injuries. Hence, they demand a careful evaluation and efficient management. Being an articular fracture, secondary degenerative changes are common and osteoarthritis may develop at a later date, however well the fracture is treated.

Mechanism of Injury

These fractures occur as a result of a direct impact onto the condyles of the tibia from the side, e.g. a vehicle striking the pedestrian. As a result of this, the knee is subjected to a severe varus or a valgus stress during axial loading. The tibial condyle is split or crushed by the opposing medial/lateral condyle of the femur.

Classification

Based on Schatzker classification—Tibial plateau fractures are classified into VI types (Figs 3.48A to F).

Clinical signs and symptoms:

a. All the signs of a fracture are present.
b. Knee may be swollen due to hemarthrosis.
c. Instability at the knee may be present.

Figures 3.48A to F

Classification of tibial plateau fractures based on Schatzker: (A) Type I—A cleavage or split; (B) Type II—A cleavage or split with depression; (C) Type III—Pure central depression; (D) Type IV—Medial condyle fracture; (E) Type V—Bicondylar fracture; (F) Type VI—Associated with metaphyseal and diaphyseal fractures.

Diagnosis

An X-ray gives the diagnosis in these fractures. Complex fractures demand detailed evaluation by CT with 3D reconstruction and MRI which reveal the exact nature of bony and soft tissue injury, respectively.

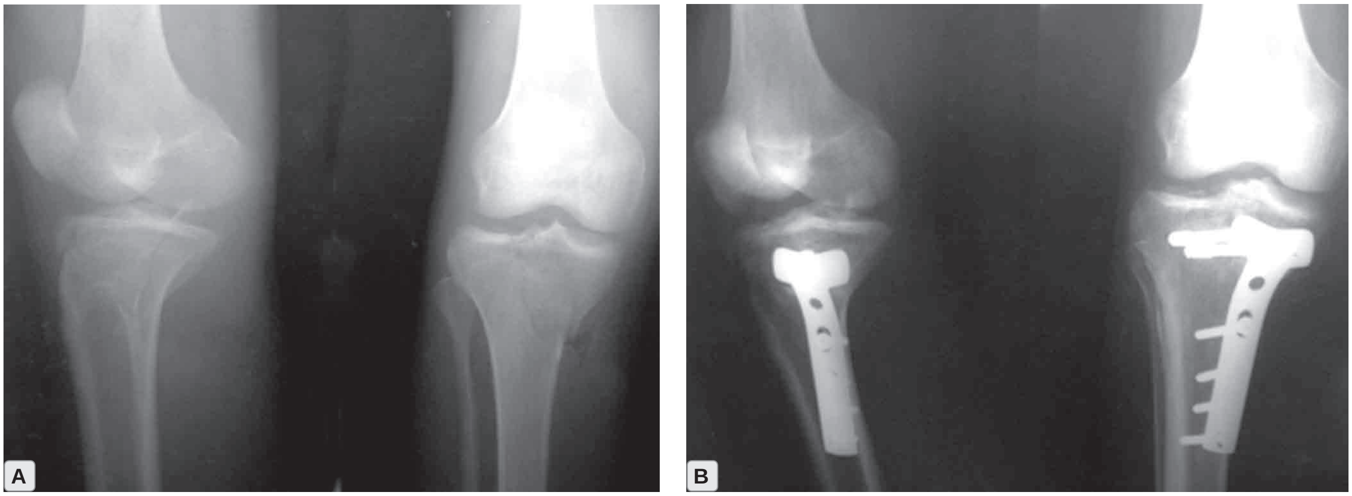
Treatment

Aim of the treatment: The treatment aims at maintaining the articular surface alignment and achieving good union at the earliest to restore the joint function. Hence, accurate reduction and fixation is required. Surgery is the treatment of choice to achieve this goal.

When defect is observed at reduction, bone grafts are used to bridge the same, especially in those fractures with depression and extensive comminution.

Nonoperative treatment:

a. *Immobilization in plaster casts:* This is instituted only in minor degrees of fractures which are undisplaced and the articular surface involvement is not extensive.
b. *Low tibial traction:* This is an excellent alternative method. It is indicated when the skin condition or the general condition of the patient does not permit surgical intervention, e.g. local area of blistering and threatened compartmental syndrome, associated head injury, etc.



Figures 3.49A and B
Schatzker Type IV fracture of the medial condyle fixed internally using a 'T' buttress plate.

Operative:

a. Closed manipulation, reduction and fixation using cannulated cancellous screws only for less severe fractures; cannulated cancellous screws along with plates and screws for more severe fractures. Plate fixation may be done under C-arm image intensifier guidance, e.g. MIPPO technique.
b. Open reduction with reconstruction of articular surface, internal fixation and repair of soft tissue injury. At times, bone grafting may be necessary (Figs 3.49A and B).

Complications

a. Compartment syndrome.
b. Injury to the popliteal vessels.
c. Injury to the lateral popliteal nerve.
d. Skin necrosis.
e. Malunion with deformity and restricted joint movement.
f. Secondary degenerative arthritis.

Essay Question

Q. Classify tibial plateau fractures. Discuss the mechanism of injury, diagnosis, management and the complications of tibial plateau fractures.

Fractures of the Femoral Condyles

Fractures involving the femoral condyles consist of:

a. Extra-articular fractures which occur just above the condyles and hence known as supracondylar.
b. Intra-articulr fractures which involve either the medial or the lateral condyle and hence known as unicondylar/condylar fractures.
c. Fractures which split the condyles and hence known as intercondylar fractures. The split may occur in the form of 'T' or 'Y'.

Mechanisms of Injury

These fractures can occur as a result of a direct injury or a fall from a height by means of the tibia being driven firmly into the condyles of the femur.

Classification (Fig. 3.50)

Based on Muller's classification, condylar fractures are divided into three groups:

Group A—Extra-articular fractures, i.e. Supracondylar fractures.

Group B—Intra-articular fractures involving one condyle, either lateral (common) or medial.

Group C—Bicondylar intra-articular fractures. These are basically intercondylar fractures with supracondylar extension. 'T' and 'Y' fractures.

Clinical Signs and Symptoms

a. All the signs of a fracture.
b. Swelling around the knee due to hemarthrosis. (If there is break in the capsule, the hemarthrosis spreads into the soft tissues)
c. All the movements at the knee are lost.

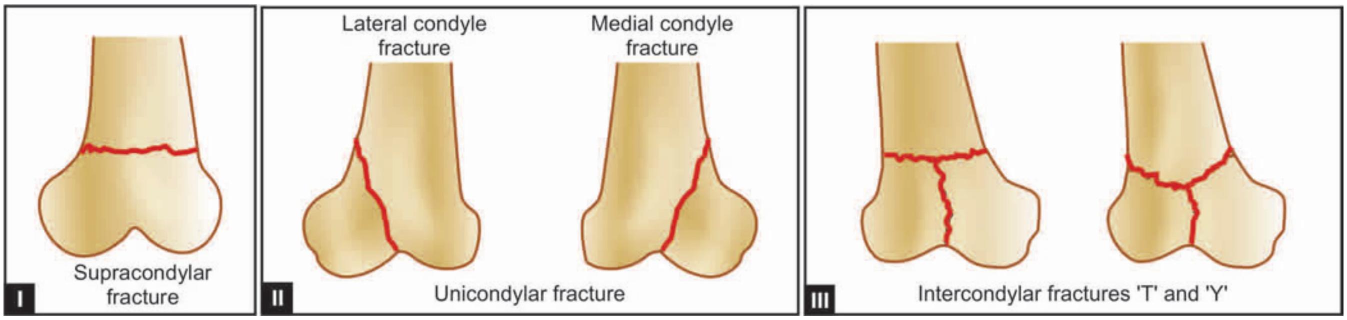
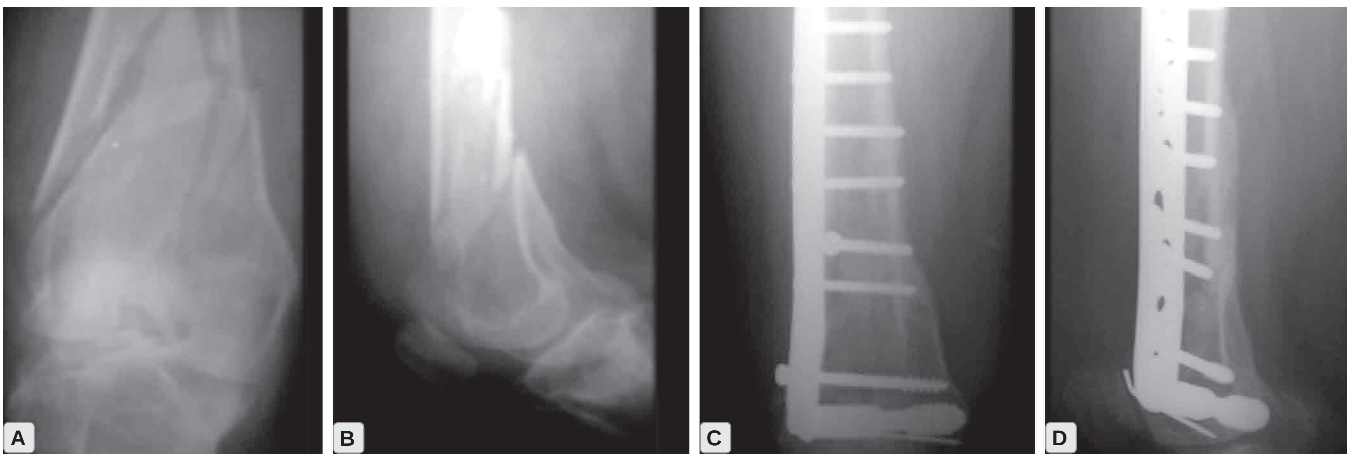


Figure 3.50

Three groups of condylar fractures based on Muller's classification.



Figures 3.51A to D

Fixation of a group C condylar fracture of the femur showing supracondylar comminution, internally fixed by means of a dynamic condylar screw. Note that the insertion of a K-wire has converted it into a supracondylar fracture. Interfragmentary screw fixation has been done for comminuted fragments in the supracondylar region. Then, the dynamic condylar screw and a Barrel plate have been inserted. At times, interfragmentary screw fixation may be necessary to hold the split condyles in position prior to the insertion of dynamic condylar screw.

Diagnosis

X-ray—Anteroposterior (AP) and lateral views give the diagnosis. Always a coronal plane fracture, i.e. the Hoffa fracture should be kept in mind.

Treatment

Though nonoperative treatment in the form of reduction and skeletal traction may result in union in less severe forms of fracture, open reduction and internal fixation is the treatment of choice. Surgery aims at achieving a sound stabilization so that early mobilization can begin to avoid disabling stiffness of the knee joint. The principle of treatment in Group C fractures is to convert it first into a supracondylar fracture and then fix it to the main fragment. When there is bad metaphyseal comminution wherein restoration of skeletal anatomy is not possible, a primary bone grafting procedure is undertaken (Figs 3.51A to D).

Essay Question

Q. Classify condylar fractures of the femur. Discuss the mechanism of injury, diagnosis management and the complications of condylar fractures.

FRACTURE OF THE SHAFT OF THE FEMUR

General Information
The fracture of the shaft of the femur is one of the major long bone fractures. The portion of the bone extending from about 7.5 cm below the lesser trochanter to about 5 cm proximal to the supracondylar portion of the femur is known as the shaft of the femur. The narrowest portion of the shaft is known as isthmus. The bone is highly vascular and surrounded all round by strong muscles of the thigh. Heavy blood loss up to 1.5 liters can occur and if not promptly replaced, patient may go in for hypovolemic shock. The accompanying soft tissue injury too is considerable and adds onto the insult.

Mechanism of Injury

A force of considerable magnitude, either bending or direct impact is necessary to break the shaft of femur. Hence, the fracture shaft femur occurs as a result of severe trauma.

Types of Fracture Femur

a. Transverse fracture
b. Oblique fracture
c. Spiral fracture
d. Comminuted fracture
e. Segmental fracture

Clinical Signs and Symptoms

The clinical diagnosis is easy as the fracture invariably gets displaced and abnormal mobility develops immediately. The thigh is swollen considerably because of heavy bleeding and associated muscle injury. Deformity and shortening of the limb is always present.

Investigations

a. Hb%, PCV and blood grouping and cross-matching is a must.
b. X-rays help in identifying the type and level of fracture as well as the diameter of the medullary canal (Proper assessment of the bone is absolutely essential before the treatment is planned).

Treatment

a. *Immediate*
   i. Immediate immobilization of the fracture in a splint and traction to prevent complications.
   ii. Administration of IV fluids and blood.
   iii. Treatment of other associated injuries if any.

Definitive treatment of the fracture is considered only after the patient is stabilized.

b. *Definitive treatment of fracture shaft femur:*
   Fracture femur in adults is always treated by reduction and internal fixation. Several methods are described.
   i. Open reduction and internal fixation using plates and screws or open nailing techniques (Figs 3.52A and B).
   ii. Closed reduction and internal fixation e.g. intramedullary interlocking nail (Figs 3.52C and D), MIPPO technique, etc.
   iii. Reduction and external fixation (Only in case of open fractures).

Closed methods are generally preferred to open methods.

Figures 3.52A to D

(A) A badly comminuted fracture of the shaft of the femur. It is not possible to achieve good apposition of fractured fragments by closed methods; (B) Showing fracture union after open reduction and internal fixation by interlocking nailing procedure; (C and D) Radiographs showing a transverse fracture mid shaft of the femur treated by closed reduction and internal fixation using an intramedullary interlocking nail. Such fractures are amenable for closed reduction and internal fixation when compared to the fracture shown in Figures A and B, which is badly comminuted and displaced.

Advantages and Disadvantages of Closed and Open Methods

Closed method

Advantages:

a. Retains the fracture hematoma and facilitates the process of union.
b. No further soft tissue damage.

Disadvantage:

a. Anatomical reduction is not possible (Acceptable alignment is possible).

Open method

Advantage:

a. Anatomical reduction is possible.

Disadvantages:

a. Fracture hematoma is lost and union depends on secondary fracture hematoma.
b. Dissection of soft tissues causes a second insult (One has to be meticulous in dissection).

Note: *Good apposition of the fragments with at least more than 75% contact is essential for union. When this is not achieved by closed methods do not hesitate to do an open reduction and internal fixation.*

Complications

Apart from routine complications like delayed union, nonunion and malunion, fracture shaft of femur poses some serious life-threatening and limb-threatening complications. They are:

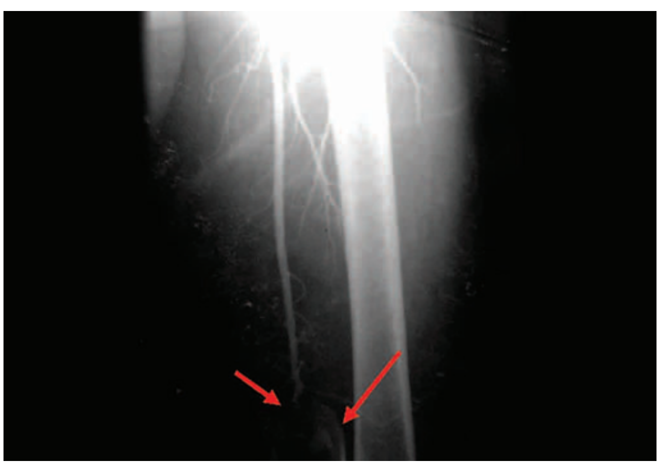


Figure 3.53

A fracture shaft femur, lower 1/3rd with an arteriogram showing transection of the femoral artery. Junction of the upper 2/3rd and lower 1/3rd is the site where the femoral artery enters the popliteal fossa through the opening in the adductor magnus muscle. This is the most vulnerable area for injury.

a. Shock and hemorrhage.
b. Fat embolism (Refer Chapter 1).
c. Injury to the femoral artery (Fig. 3.53).
d. Injury to the sciatic nerve.

These complications have to be identified and dealt with efficiently to avoid a disastrous outcome.

Essay Question

Q. Discuss the mechanism of injury, diagnosis, management and complications in femoral shaft fractures.

FRACTURES AROUND THE HIP

The fractures seen around the hip are:

a. Fracture of the head of the femur (Very very rare. Usually associated with hip dislocation).
b. Fracture of the neck of the femur (True intracapsular fracture).
c. Intertrochanteric fracture (True extracapsular fracture).
d. Subtrochanteric fracture (Fractures occurring below the lesser trochanter).

Fracture of the Neck of the Femur

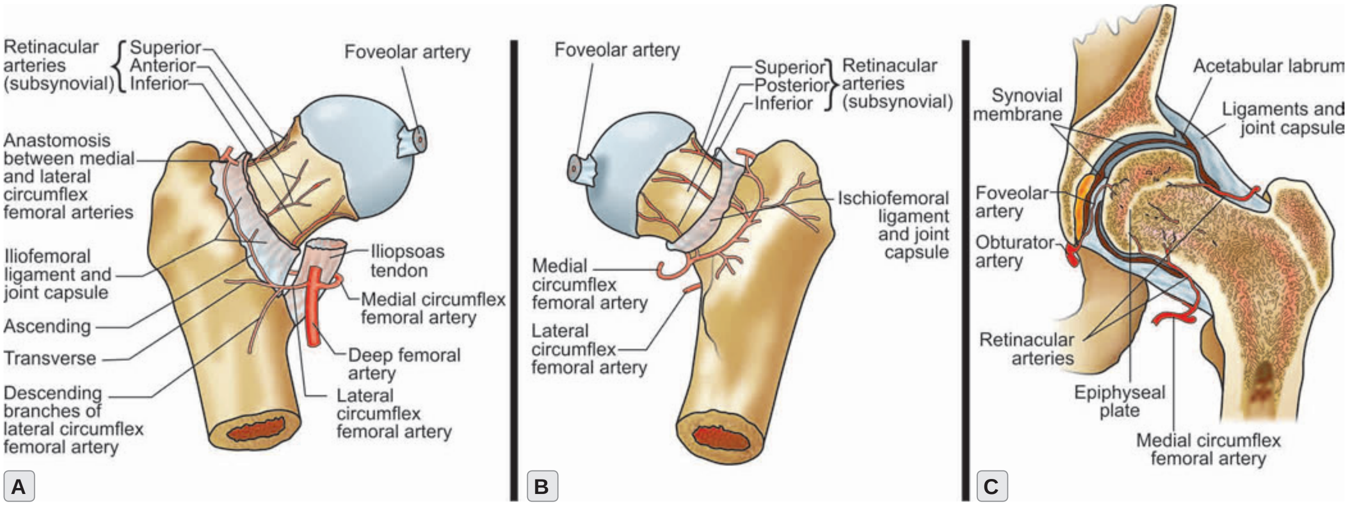
General Information

This fracture commonly occurs in elderly in the 7th decade and in women with osteoporosis (after menopause), following a very trivial torsional trauma (slipping). Sir Astley Cooper was convinced about this fact way back in 1800's and said "A patient sustains a fracture and then falls and not that he falls and then sustains a fracture." Though this is true in elderly patients, in the young more severe trauma is necessary to produce a fracture neck of the femur. Thus, it is natural to conclude that more than one mechanism operates to cause a fracture neck of the femur.

This fracture even today poses several problems to the treating surgeon. Hence, is known as a problem fracture, unsolved fracture and some call this (out of frustration) as an unsolvable fracture.

Problems Associated with the Fracture

1. There is no role for nonoperative treatment. Surgery is the treatment of choice.
   Whitman spica, given in the yesteryears was a failure and also associated with all the problems of prolonged confinement to bed, e.g. bedsore, hypostatic pneumonia, deep vein thrombosis, etc.
2. Difficulty encountered at reduction because of the spherical nature of the head which when broken,



Figures 3.54A to C

Blood supply to the head and neck of the femur. From the anastomotic vascular ring formed at the base of the neck by the medial and the lateral circumflex femoral vessels, arise the anterior and posterior retinacular vessels which run along the neck of the femur towards the head and supply it. Also note area supplied by the foveolar artery which is very negligible when compared to the area supplied by the retinacular vessels. When the neck is fractured, the retinacular vessels are invariably damaged. More so, if the fracture is displaced: (A) Anterior view; (B) Posterior view; (C) Cross-section view.

is free to rotate within the acetabulum (To achieve reduction, a fracture table is necessary and different methods of reduction are to be employed).

3. No design of the implant, currently available satisfies all the criteria necessary for a secure fixation of the proximal fragment (This is true because of the small size and spherical shape of the proximal fragment. The entire variety of implants designed so far from Smith Peterson nail, Moores pins, Knoweles pins, Deyreles pins, Asnis screws, Garden screws to cannulated cancellous screws justifies this fact).
4. Precarious blood supply may cause avascular necrosis of the head and nonunion of the fracture. (Retinacular vessels run along the neck of the femur. These retinacular vessels break when the neck is fractured. Artery through the ligament of the head of the femur supplies approximately 10% of the head) (Figs 3.54A to C).
5. The fracture being intracapsular, the synovial fluid interferes with the stages of healing.

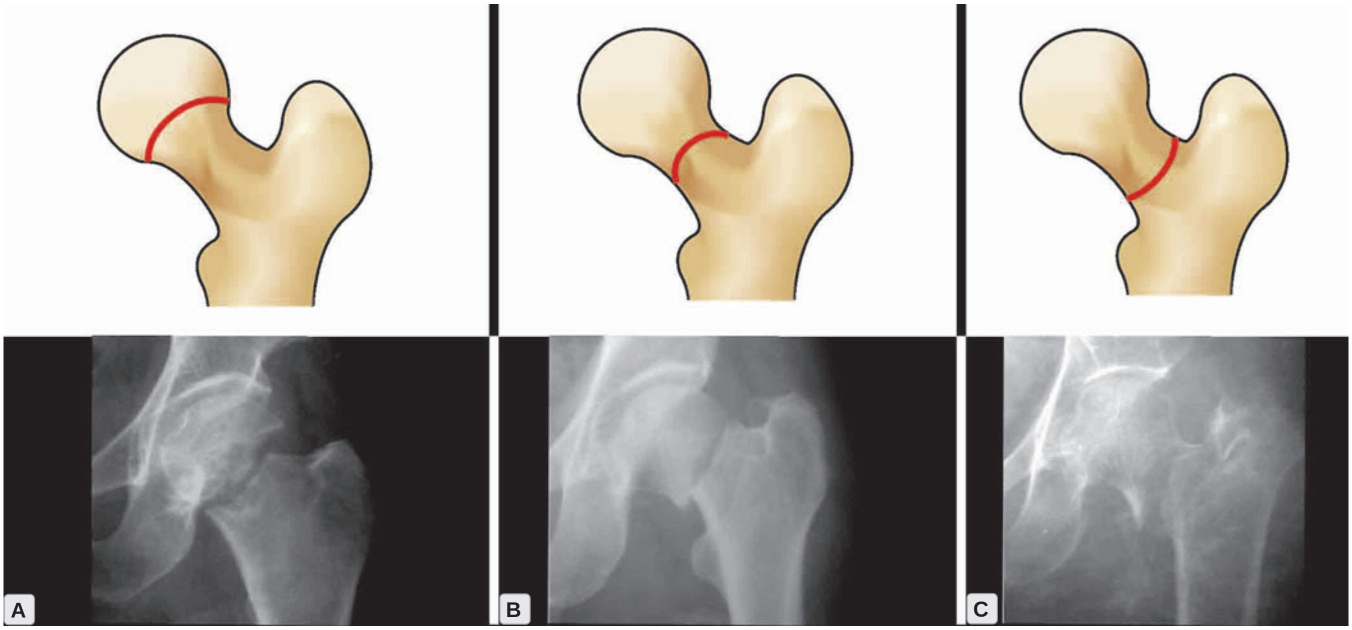
Problems of Old Age Associated with the Fracture

During old age, a person is susceptible for multiorgan failure and perhaps lives with compromised vital organs. Thus the diseases of old age like diabetes, hypertension, central nervous system (CNS) compromise like stroke, renal compromise, poor lung function, etc. all complicate the ensuing surgical procedure. The incidence of deep vein thrombosis (DVT) also increases with advancing age.

Because of the above reasons, the names problem, unsolved and unsolvable fracture are fully justified even today.

Mechanisms of Injury

a. *Indirect mechanism (torsional stress, low energy trauma):* This is the most common mechanism operating in elderly patients when they slip and the limb rotates externally. It is postulated that if the hip is in extension, the head of the femur remains fixed in the acetabulum so that the external rotation stress of the slip, falls on the neck of the femur and breaks it. More distal fractures occur if the hip is in 5–10° flexion, during the external rotation stress.
b. *Direct mechanism:* Here, as a result of a fall on the trochanter, the force is transmitted to the neck directly and the neck breaks.
c. *Axial loading (high energy trauma):* The force is transmitted along the long axis of the femur with or without rotational component. This is the most common mechanism operating in the young.



Figures 3.55A to C

Different types of fractures of the neck of femur: (A) Subcapital; (B) Transcervical; (C) Basal.

Classification

Depending on the site (Figs 3.55A to C)

A. *Subcapital* (Constitutes about 70% of the fractures. Fractures occur just below the caput or the head of the femur/at the site where the epiphyseal plate existed)

B. *Transcervical* (Constitutes about 20% of the fractures. Fractures occur through the neck of the femur)

C. *Basal* (Constitutes about 10% of the fractures. Fractures occur at the base/origin of the neck)

Depending on the displacements

IV stages are identified based on Garden's classification depending on the displacements.

Garden's classification (Figs 3.56 and 3.57)

Stage I—Incomplete fracture (or valgus impaction)

Stage II—Complete fracture without displacement

Stage III—Complete fracture with minimal displacement

Stage IV—Complete fracture with marked displacement

Clinical Signs and Symptoms

1. Following trivial/severe trauma, a patient develops severe pain in the hip and inability to stand and walk.
2. The involved limb is in flexion, abduction and external rotation *(Helpless attitude of the* limb).
3. Minimal shortening is observed.
4. There is tenderness over the mid inguinal point.
5. The greater trochanter is normal and non-tender.

Diagnosis

Radiography: Views suggested are X-ray pelvis with both hips AP with the limb in maximum internal rotation (to visualize the neck and to assess the quality of the bone) and cross-table lateral view (to visualize the anterior and posterior portion of the neck as well as posterior comminution).

Bone scan and MRI in late presentations are useful to diagnose AVN.

Bigelow's dictate:* In 1864, Bigelow remarked that *"While the impacted fracture of the base of the femoral neck unites by bone, if at all, there seems to be a decreasing tendency to osseous union as we approach the smaller portion of the neck near its head."

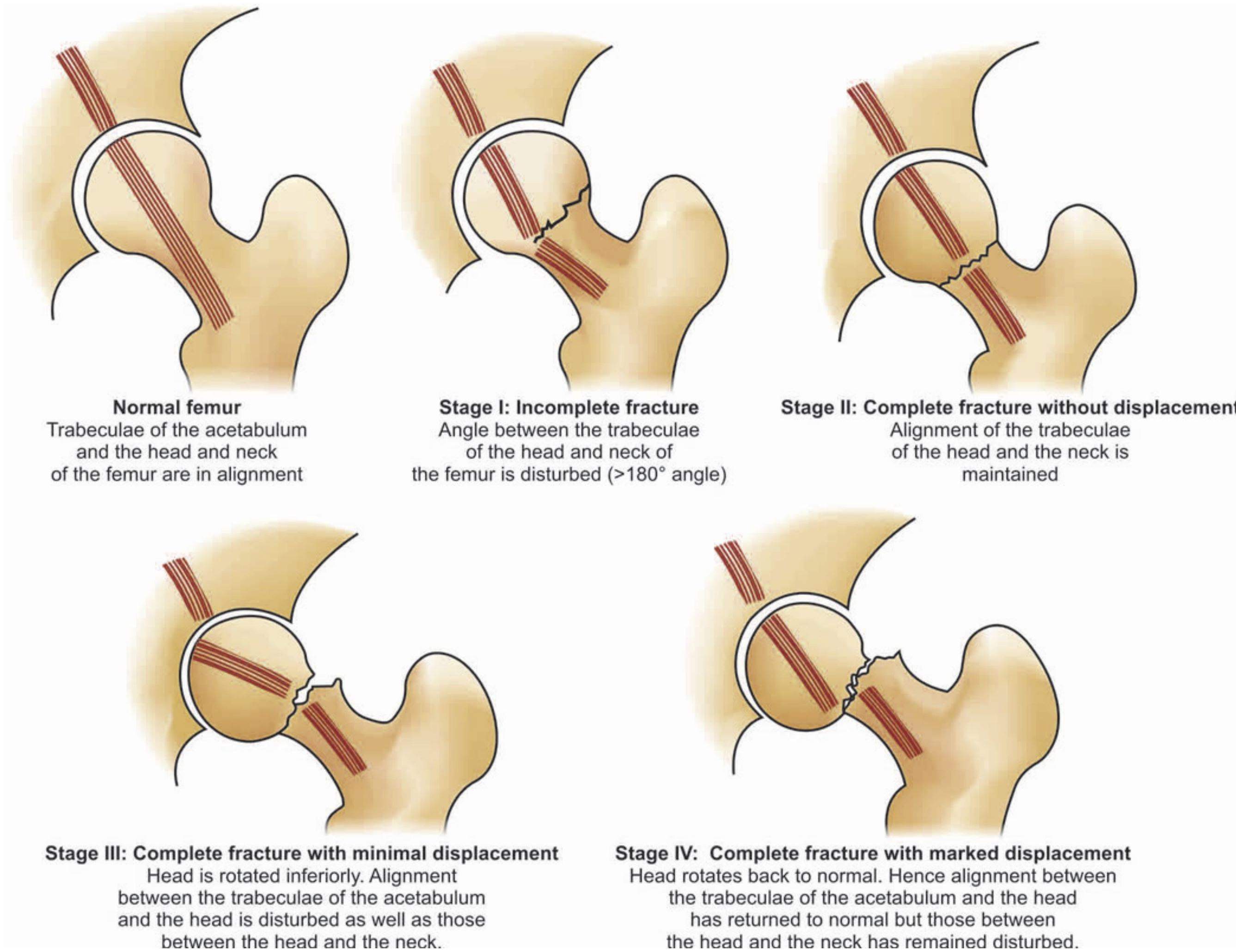
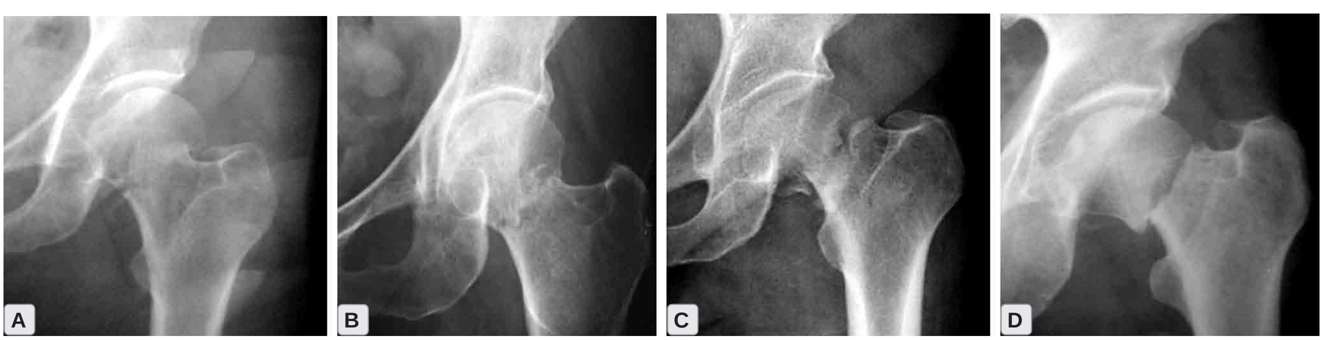


Figure 3.56

Trabecular pattern along the head and neck of the femur in different types of fractures. The fracture displacement is evaluated by looking at the relation of the trabecular pattern of the neck with the shaft for Stage I and Stage II fractures and trabecular pattern of the neck with the acetabulum for Stage III and Stage IV fractures. Break in continuity and loss of relation of the trabecular pattern is observed specifically in different stages.



Figures 3.57A to D

All the four stages of fracture neck of femur based on Garden's classification. Note the break and trabecular disruption between the head and neck of the femur as well as the acetabulum. In Stages I and II the trabecular pattern in the head and neck is taken into consideration. Stages III and IV trabecular pattern between the head and acetabulum is taken into consideration.

Management

All fractures are managed by surgery.

Note: *Impacted fracture can be managed by supported weight bearing after an initial period of rest for 2–3 weeks. In situ fixation is the other method practised for treating impacted fracture*.

Surgical procedures employed are:

a. Internal fixation.
b. Arthroplasty (Fig. 3.58).
c. Reconstructive procedures (Indicated in old fractures with complications).

Factors to be considered before internal fixation:

a. *Age of the patient*: At a younger age, attempt should always be made to preserve the head and neck of the femur. Hence, the fractures are to be anatomically reduced and internally fixed. (Closed reduction and internal fixation. The fracture site is not opened).
b. *Type and nature of the fracture:* Displaced and comminuted fractures indicate severe trauma and are more prone for complications of AVN and nonunion even after good internal fixation. Hence, such fractures are to be treated at the earliest to minimize the risk of AVN and nonunion.
c. *Duration of the fracture:* Longer the duration of the fracture, the incidence of AVN and nonunion is more. Hence, the fixation is to be augmented with vascularized bone grafting techniques, e.g. Meyers muscle pedicle graft.
d. *Quality of bone* (*Singh index*): The quality of the bone has a direct relation to quality of healing. Osteoporotic bone of an old person heals poorly when compared to a nonosteoporotic bone of the young.

Implants used for internal fixation: Cannulated cancellous screws, Garden screws, Moore's pins and Knowle's pins.

Factors to be considered before replacement hemi-arthroplasty

a. *Age of the patient:* Elderly whose activity as well as life expectancy is limited and who cannot tolerate prolonged confinement to bed till the fracture unites are considered for arthroplasty.
b. *Type of the fracture*: All fractures other than incomplete fractures and impacted fractures are suitable for arthroplasty.
c. *Duration of the fracture:* Duration has no relation to arthroplastic procedures. It can be done at any period of presentation after good preoperative evaluation with appropriate prosthesis.

   When calcar femorale is sufficient, Austin Moore's prosthesis is used. When calcar femorale is insufficient, Thomson's prosthesis is used with bone cement.

   (Cemented bipolar prosthesis and primary total hip arthroplasty are also the treatment modalities to be considered in selected cases).

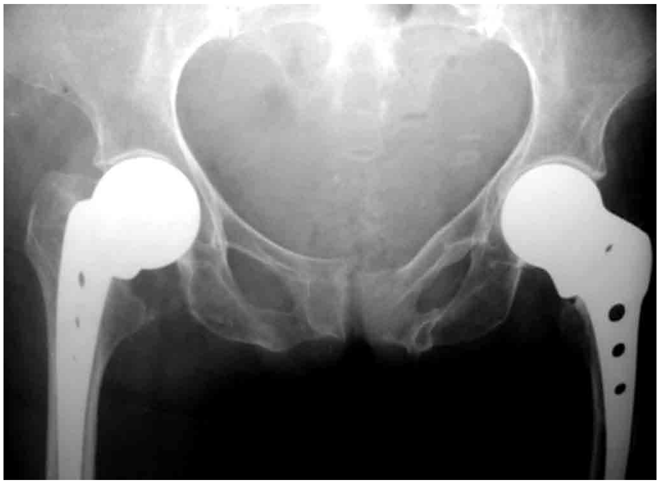


Figure 3.58

Bilateral hemiarthroplasty using Austin Moore's prosthesis done for a case of bilateral fracture neck femur. First the patient sustained the fracture in the right hip and an year later in the left hip.

Treatment modalities depending on status and vascularity of the neck and head of the femur:

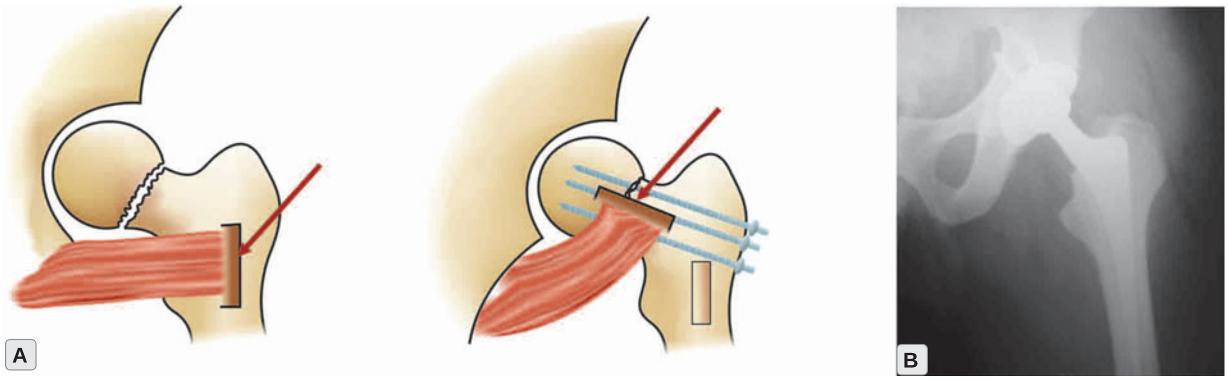
Management of AVN with or without nonunion:

a. In the older age group, cemented total joint arthroplasty is the procedure of choice.
b. In the young, uncemented total joint arthroplasty is the procedure advocated.

Management of nonunion in young without AVN:

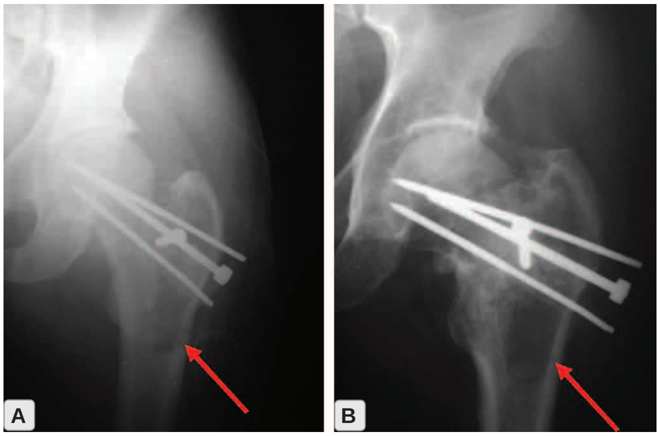
a. Without resorption of the neck of the femur—reconstructive procedure in the form of vascularized muscle pedicle graft (Figs 3.59A , and 3.60A and B).
b. With resorption of the neck of the femur—uncemented total hip arthroplasty (Fig. 3.59B).

To conclude fracture neck of the femur is treated by different methods considering several factors. The Flow chart 3.1 summarizes the different methods employed.



Figures 3.59A and B

(A) Quadratus femoris vascularized muscle pedicle grafting procedure and; (B) An uncemented total hip replacement.



Figures 3.60A and B

Quadratus femoris muscle pedicle bone grafting procedure done for a 3-month-old ununited fracture neck of femur without AVN and resorption of the neck; (A) Immediate postoperative X-ray; (B) X-ray at 3 months follow-up. Note that the fracture has soundly united and there is no evidence of AVN during follow up. The arrow points out at the resultant defect at the donor site which gradually gets filled up with bone.

Relationship between Senile Osteoporosis and Fracture Neck of Femur

1. It acts as a predisposing factor for a fracture to occur with a trivial trauma (Not seen among African Americans and Bantu race in whom osteoporosis is uncommon).
2. It is responsible for comminution of the neck.
3. It is responsible for poor healing.
4. It does not allow firm purchase of the implant in the bone, causes loosening of the implant and failure of fracture fixation.

'Singh Index of Osteoporosis' and its Importance in Fracture Neck of Femur

Singh Index (For more information Ref: Singh. et al JBJS 1970;52-A:457-67).

This index is based on the nature of the bony trabeculae of the proximal femur. They are grouped as follows:

a. Ward's triangle.
b. Greater trochanteric Group.
c. Secondary compressive Group.
d. Secondary tensile group.
e. Principal tensile group.
f. Principal compressive group.

Depending on the texture they were assessed and graded into VI Grades, Grade VI being normal and Grade I suggests severe osteoporosis.

Grade VI
- All normal trabecular groups are visible.
- Upper end of femur seems to be completely occupied by cancellous bone.

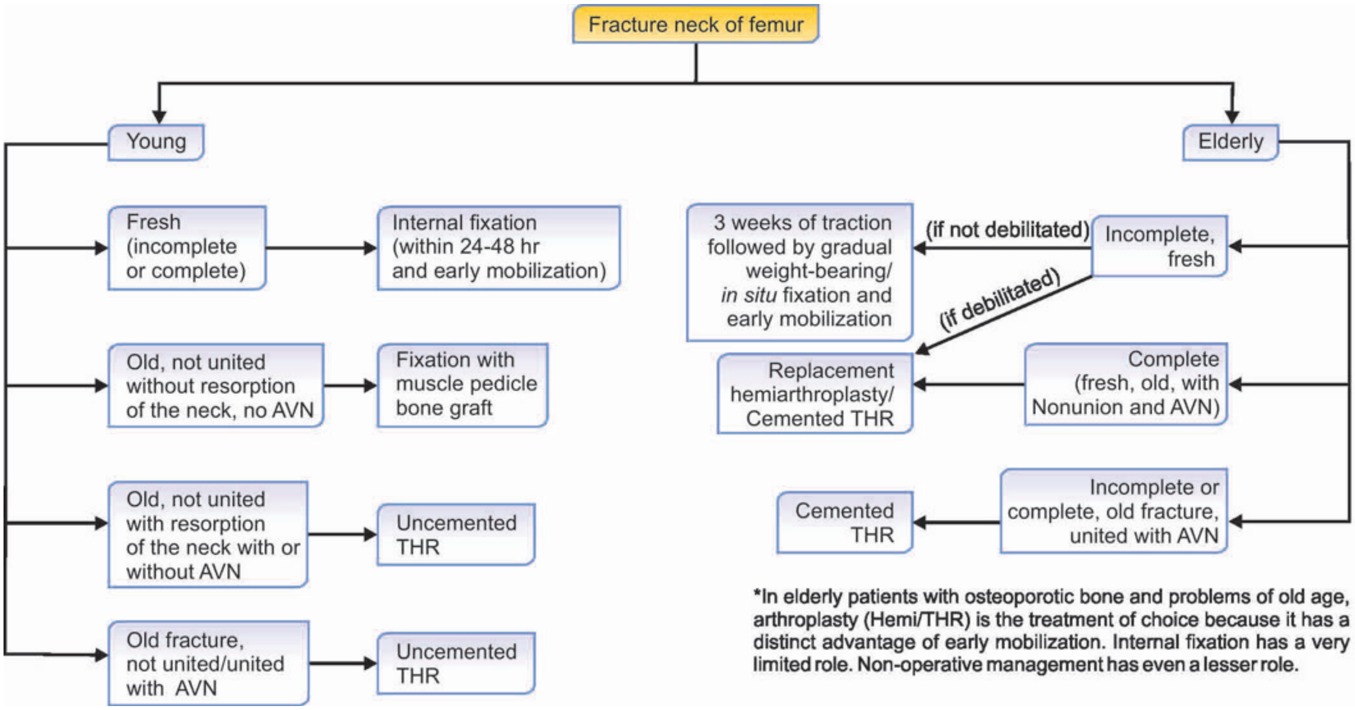
Grade V
- Principal tensile and principal compressive trabeculae is accentuated *(thinned out).*
- Ward's triangle appears prominent.

Grade IV
- Principal tensile trabeculae are markedly reduced but can still be traced from lateral cortex to upper part of the femoral neck. *(Thinned out and reduced but no break is observed).*

Grade III
- There is a break in the continuity of the principal tensile trabeculae opposite the greater trochanter *(break in continuity observed).*

Flow chart 3.1

Treatment plan for fracture neck of the femur.



Grade II
- Only principal compressive trabeculae stand out prominently.
- Remaining trabeculae have been essentially absorbed *(absence of tensile trabeculae and thinning of compression trabeculae)*.

Grade I
- Principal compressive trabeculae are markedly reduced in number and are no longer prominent *(few compressive trabeculae are left)*.

Note: *Grade III -I are indicators of severe Osteoporosis, Grade- I being very severe.*

Singh index helps in the fixation of fracture neck of femur in the following way:

1. It gives the physiological age of the bone (physiological age is different from chronological age).
2. Helps in taking a decision whether to fix or not to fix. Grade VI to Grade IV are acceptable indices for fixation. Grade III to Grade I are poor indices for fixation.
3. Helps in placement of the implant in the non-osteoporotic inferior part of the neck (low insertion to prevent cut through).

Relationship of Garden's Alignment Index to Fracture Neck of Femur

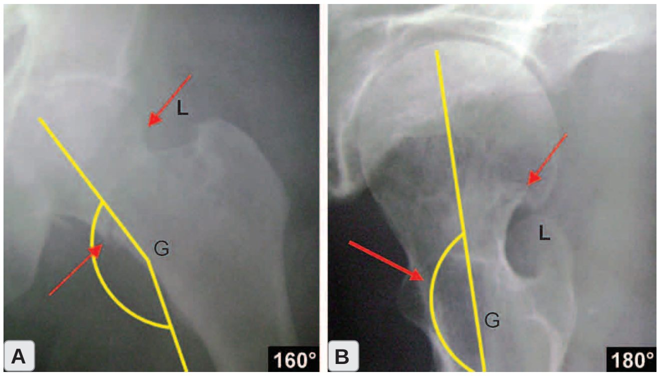
Garden's alignment index: It is an index derived from two angles.

i. Angle formed by the primary compression trabeculae with the longitudinal axis of the femoral shaft in AP view
ii. Angle formed by the primary compression trabeculae with the longitudinal axis of the femoral shaft in lateral view.

The ideal index is 160°/180° i.e. 160° in the AP view and 180° in the lateral view (Figs 3.61A and B) with a variation of + or −10°. This index helps in assessing the reduction of the fracture as acceptable when the angle is within this range. < 155° and > 180° increases the risk of AVN by almost 50%. Nonunion is also commonly seen when the angle of reduction is beyond this acceptable range.

Lowell's Alignment Theory

Radiographically, the outline of the femoral head is convex and the outline of the femoral neck is concave.



Figures 3.61A and B

AP and lateral views of hip joint showing satisfactory reduction as per Garden's alignment index. Also see the Lowell 's reverse 'S' curve. * Garden's alignment index-G; Lowell's alignment theory-L Refer text for details.

Together they form an S (right hip) or a reversed S (left hip) shaped curve. Instead of S, if an unbroken C curve is seen after reduction, the reduction is considered as unacceptable. Fig. 3.61A at the place where the arrow is pointing, the curve is reverse 'S' shaped and smooth suggesting acceptable reduction. On the right side the curve formed would take the shape of an 'S'.

Revision Questions

- Q. Classify fracture neck of femur.
- Q. Discuss complications of fracture neck of femur.
- Q. Garden's alignment index.
- Q. Singh index.

Essay Questions

- Q. Discuss the mechanism of injury, classification, diagnosis and management in fracture neck of femur.
- Q. Fracture neck of femur an unsolved fracture. Discuss.
- Q. Enumerate the complications in fracture neck of femur. Discuss their management.

Intertrochanteric Fracture

Intertrochanteric fractures are those fractures occurring in the extracapsular portion of the proximal femur, i.e. in the portion between the greater and the lesser trochanter of the femur. These fractures, like fracture of the neck of the femur, occur in elderly people in the later part of the 7th decade and in 8th decade, i.e. 5–10 years later than fracture neck of the femur. Fractures occur beyond the attachment of the capsule of the hip joint. The bone in this region is cancellous in nature and hence the union rate in these fractures is higher when compared to fracture neck of femur. Also, these fractures are associated with less complications.

Note: *The capsule of the hip joint is attached anteriorly at the intertrochanteric line and posteriorly stops short about a centimeter before the intertrochanteric crest.*

Mechanisms of Injury

a. *Indirect mechanism (torsional stress, low energy trauma):* It is postulated that a torsional stress occurring in an elderly individual with osteoporotic femur can cause this fracture. The hip should be in some degree of flexion for the torsional force to act on the intertrochanteric portion of the femur.

b. *Direct mechanism:* A direct impact on the trochanter resulting in intertrochanteric fracture.

c. *Axial loading (High energy trauma):* Commonly seen in young patients, e.g. vehicular accidents, fall from a height.

Classifications

Based on Evans' classification (1949): Classified as follows (Figs 3.62A to F)

Type I—Undisplaced 2-fragment fracture.

Type II—Displaced 2-fragment fracture.

Type III—3-fragment fracture without posterolateral support.

Type IV—3-fragment fracture without medial support.

Type V—4-fragment fracture without posterolateral and medial support.

Type VI—Reverse oblique fracture.

(Type I is a stable fracture. When Types II, III, IV and V remain displaced after attempted reduction, they are considered unstable. When reduced satisfactorily they are considered stable. Type VI—Always unstable).

Based on Boyd and Griffin's classification (1949): Classified as follows (Figs 3.63 and 3.64)

Type I—Linear intertrochanteric fractures (without comminution).

Type II—Intertrochanteric fractures with comminution.

Type III—Intertrochanteric fracture with subtrochanteric extension.

Type IV—Fractures of the trochanteric region and the proximal shaft (fracture in two planes).

Clinical Signs and Symptoms

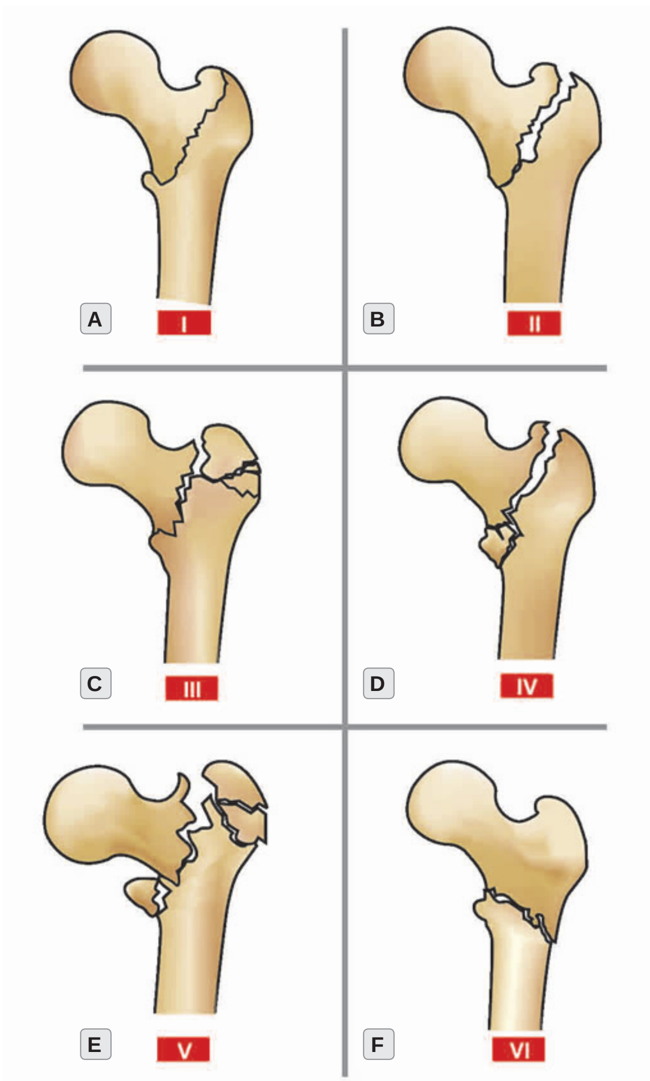
Following history of a trivial trauma in elderly and severe trauma in young

1. Gross shortening of limb.
2. Flexion, abduction and, external rotation deformity.
3. Tenderness and irregularity over the greater trochanter.

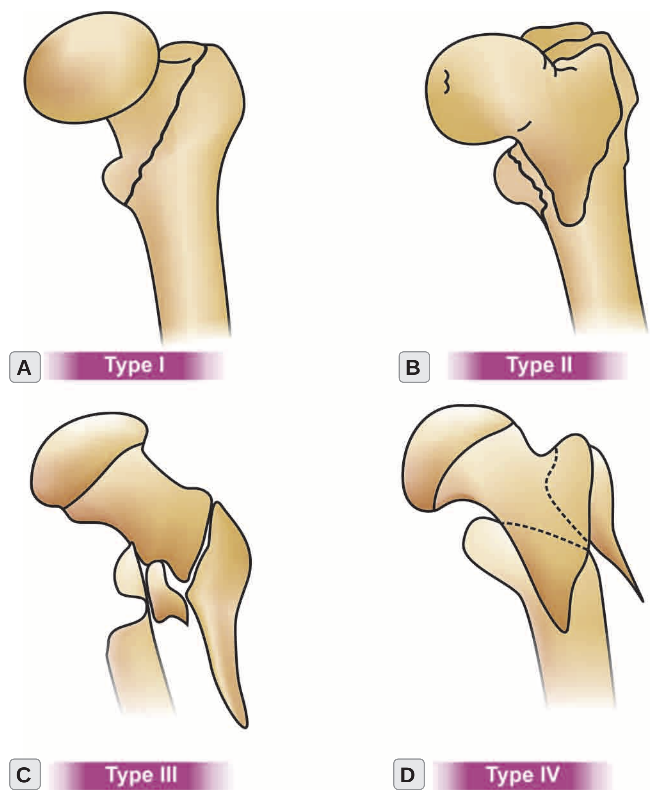
Following are the differences in clinical features between intertrochanteric fracture and fracture neck of femur (Table 3.1).

Table 3.1: Differences between intertrochanteric fracture and fracture neck of femur

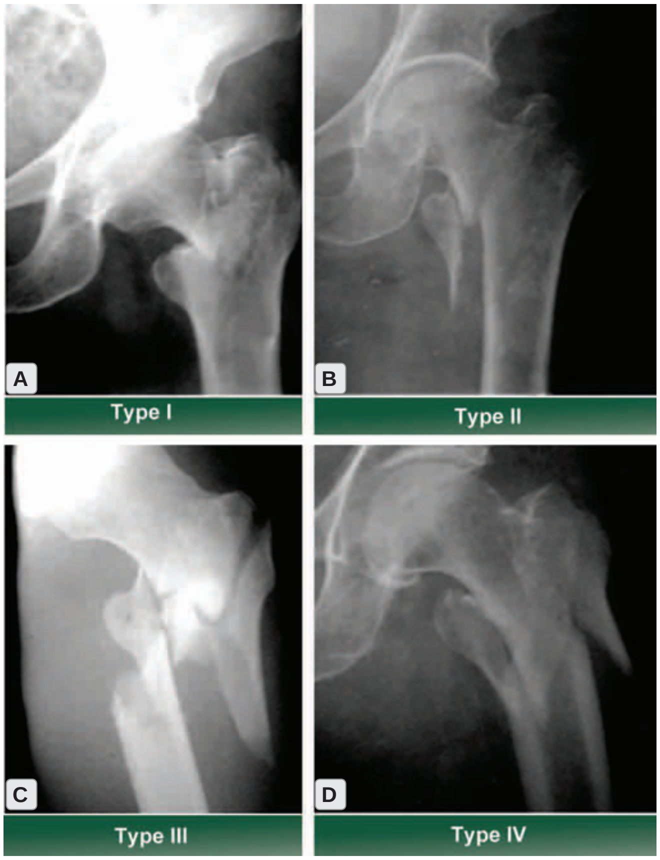
Differences	*Intertrochanteric fracture*	*Fracture neck of femur*
Attitude of the limb	Marked external rotation	Minimal external rotation
Shortening	Marked	Minimal
Tenderness	Over the trochanter	Over the mid inguinal point
Irregularity	Over the trochanter	Trochanter is normal
Swelling	Considerable, in the trochanteric region and in the upper 1/3rd thigh	Minimal, around the hip only



Figures 3.62A to F
Evans classification of intertrochanteric fracture.



Figures 3.63A to D
Boyd and Griffin classification of intertrochanteric fracture.

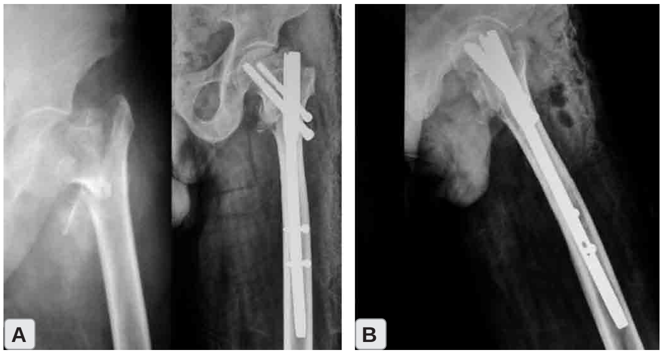


Figures 3.64A to D

Intertrochanteric fractures. Radiographs showing four types of fractures based on Boyd and Griffin classification.

Figure 3.65

Radiograph showing union of an intertrochanteric fracture after dynamic hip screw fixation. Note the central placement of the screw in the head and neck of the femur in the AP view. The same is desirable in lateral view too for a stable fixation.



Figures 3.66A and B

(A) AP view of Type II Boyd and Griffin intertrochanteric fracture and (B) showing radiograph aftrer fixing (CRIF) with proximal femoral nail (PFN).

Diagnosis

By means of standard AP view of the pelvis with both hips and cross table lateral view.

Management

a. *Operative:* Results are far superior to nonoperative methods and fractures invariably unite when fixed by proper techniques using appropriate implants. Risks of surgery in elderly patients and poor quality of bone, do pose problems for operative management.
   Stable intertrochanteric fractures—The fracture is reduced on a fracture table and internal fixation is done using a Sliding hip screw and a Barrel plate (Fig. 3.65). Failure to reduce by closed reduction on a fracture table, necessitates open reduction of the fracture before internal fixation.
   Unstable intertrochanteric fractures—Medial arch involvement with fracture of the calcar and posterior comminution are the causes for instability. These have to be well reduced and internally fixed by providing additional support. Buttress plate, trochanteric hook plate, interfragmentary screws are some of the additional implants that are useful. Proximal femoral nail (PFN—Figs 3.66A and B) can be used in selected cases. Modified internal fixation techniques are employed in certain fractures e.g. Dimon-Houston technique, Sarmiento technique, etc.
b. *Nonoperative:* Not desirable in elderly as chances of succumbing to problems of prolonged confinement to bed are very high.

Nonoperatively managed by upper tibial skeletal traction on a supportive splint for 6–8 weeks with appropriate weights.

Factors affecting union:
1. Poor quality of bone.
2. Poor quality of muscles.
3. Unstable fractures.
4. Inappropriate technique of fixation.

Essay Question

Q. Discuss the mechanisms of injury, classifications, diagnosis and management in intertrochanteric fracture of femur.

Subtrochanteric Fracture

The part of the bone from the lower end of the lesser trochanter to about 5–7.5 cms below the lesser trochanter (depending on the height of the individual), is considered as the subtrochanteric portion of the femur. A fracture occurring in this portion of the femur is known as subtrochanteric fracture.

These fractures are considered to be one among the most difficult fractures to treat for following reasons.

i. The bone involved is cortical. Hence, it takes a longer time to heal when compared to a cancellous bone.

ii. This area is subjected to greater stress when compared to rest of the femur. Hence, failure of treatment is common.

Mechanisms of Injury

a. Direct—a direct impact onto the lateral thigh by a fall. This mechanism is common in elderly.

b. High velocity injury, e.g. road traffic accident. This mechanism is common in the young wherein a force of considerable magnitude breaks this portion of the bone.

Note: *Subtrochanteric region, is one of the areas which is subjected to maximum stress during activities of daily living. Hence, the bone is quite strong and does not break easily.*

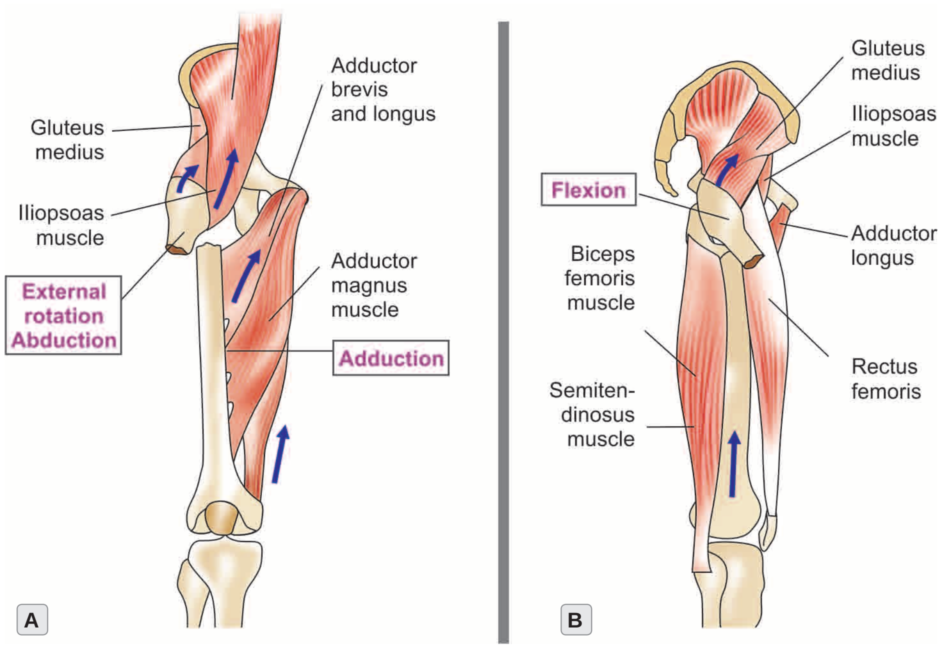
Following fracture, the muscles attached to the proximal femur displace the fractured fragments (Figs 3.67A and B). The proximal fragment is in flexion and abduction and the distal fragment is in adduction because:

a. The gluteus medius and minimus abduct the greater trochanteric (proximal) segment.

b. The iliopsoas flexes the lesser trochanteric (proximal) segment.

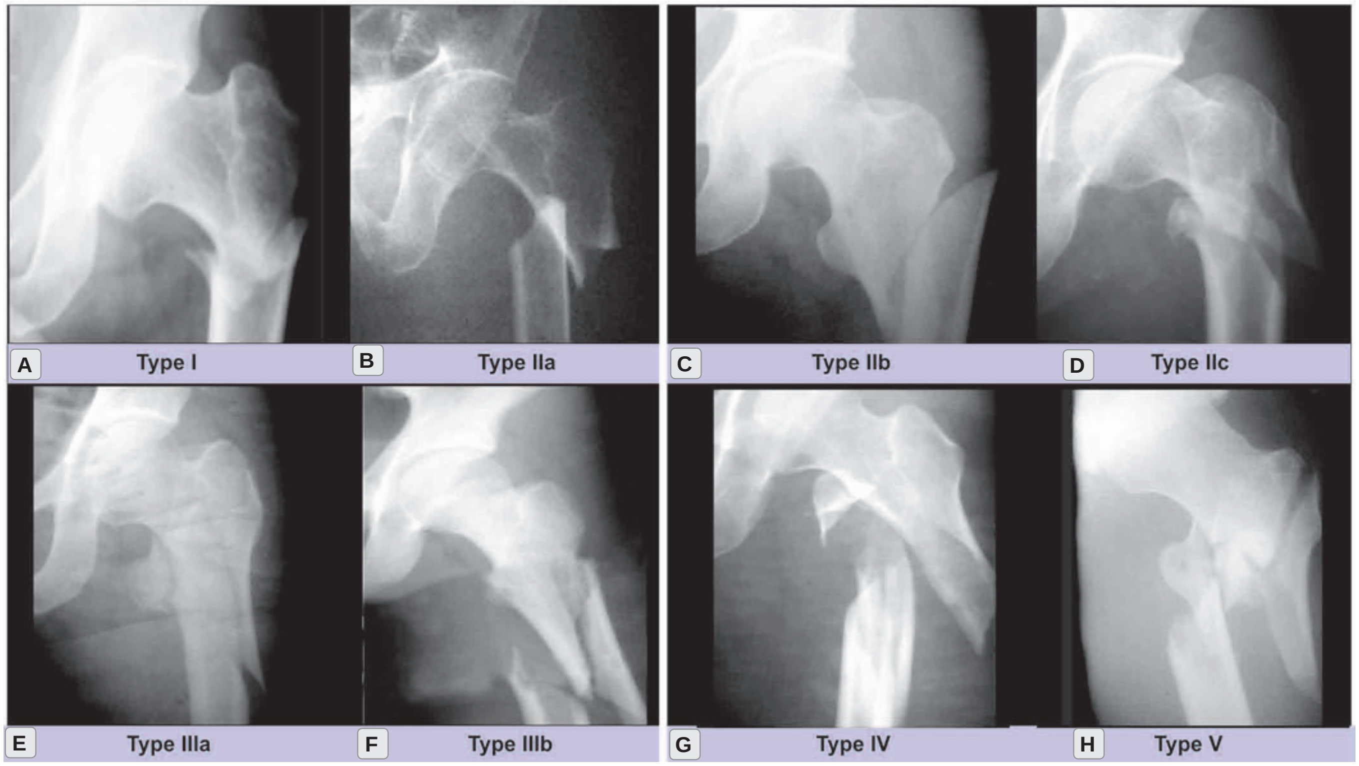
c. The adductors, adduct the shaft (distal) segment.

As this area is highly vascular heavy bleeding also is common following a fracture.



Figures 3.67A and B

Displacing forces in a subtrochanteric fracture: (A) Anterior view; (B) Lateral view.



Figures 3.68A to H

Subtrochanteric fractures—based on Seinsheimer classification. Only Type I is non-displaced fracture. Type II, III,IV and V are displaced fractures. Refer text for details.

Classification

Based on Seinsheimer classification (Ref: J Bone and Joint Surgery Am 1978;60:300-6) (Figs 3.68A to H).

Type I Nondisplaced fractures/fractures < 2 mm displacement.

Type II Two-part fracture.

A. Transverse fracture.

B. Spiral fracture with lesser trochanter in the proximal fragment.

C. Spiral fracture with lesser trochanter in the distal fragment.

Type III Three-part fracture.

A. With lesser trochanter as separate third fragment.

B. The third part is a butterfly fragment other than lesser trochanter.

Type IV Four-part (or more) comminuted fractures.

Type V Subtrochanteric with intertrochanteric fracture (Fracture through the greater trochanter).

Treatment

Nonoperative: There is no role for nonoperative treatment unless the patient is not fit for surgical procedure.

The non-operative treatment consists of skeletal traction on a supportive splint which is maintained till the fracture unites.

Operative: Surgical stabilization is the treatment of choice.

Implants used currently are cephalomedullary and centromedullary interlocking nails.

Surface implants such as locking compression plates, dynamic hip screw, dynamic condylar screw, etc. are also used in selected cases. As far as the decision with respect to choice of the implant required for the fixation of subtrochanteric fracture, two major variables are to be considered:

- Whether the fracture is extending into the greater trochanter posteriorly and involving the piriformis fossa. (Because it is one of the most commonly used nail entry portal).
- Whether there is continuity of the lesser trochanter.

(Refer Russell–Taylor classification for guidance in selecting the appropriate implant)

Implants used for internal fixation in fracture around the hip

a. Fracture neck of femur
   i. Moore's pin
   ii. Knowle's pin
   iii. Garden hip screw
   iv. Asnis' screw
   v. Cannulated cancellous screw
b. Intertrochanteric fracture
   i. Richard's screw and barrel plate
   ii. Dynamic hip screw and barrel plate
   iii. Proximal femoral nails.
c. Subtrochanteric fracture
   i. Dynamic hip screw and barrel plate
   ii. Proximal femoral nail
   iii. Reconstruction nail

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4 Dislocations

- *Specific dislocations in the upper limb, their clinical features, diagnosis and management*
- *Specific dislocations in the lower limb, their clinical features, diagnosis and management*

DISLOCATION OF THE ACROMIOCLAVICULAR JOINT

General Information
The acromioclavicular joint is a diarthrodial joint between the lateral end of clavicle and the acromion. A fibrocartilaginous disk is interposed between the two articular surfaces preventing them from coming into direct contact. The stability of the joint depends mainly on the capsule and the ligaments binding the joint.

The acromioclavicular joint dislocates only when the coracoclavicular and the acromioclavicular ligaments are completely torn.

Mechanism of Injury (Fig. 4.1)

Fall on the tip of an adducted shoulder first causes a strain on the acromioclavicular ligaments which results in subluxation of the acromioclavicular joint. If the force continues to act further, it ruptures the conoid and trapezoid part of the coracoclavicular ligament and a complete dislocation of the acromioclavicular joint occurs.

Classification (Fig. 4.2)

The injury is classified into 3 types as follows.

Type-I: It is an acute sprain (contusion) of the acromioclavicular ligaments.

Type-II: Rupture of the acromioclavicular ligament occurs.

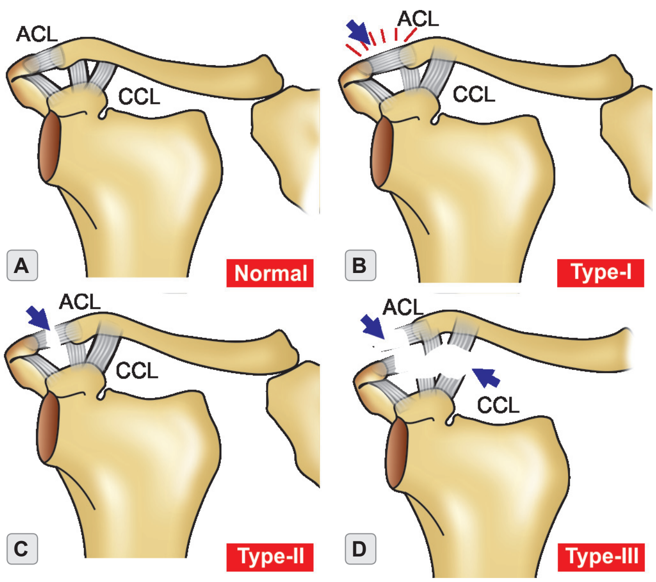
Type-III: Rupture of both acromioclavicular and the coracoclavicular ligament occurs.

In Type-I, there is no joint subluxation or dislocation. In Type-II, there is subluxation of the acromioclavicular joint. In Type-III, there is dislocation of the acromioclavicular joint.

Figure 4.1
Most common mechanism of injury of acromioclavicular joint which is fall on the tip of adducted shoulder.

Clinical Features

a. Pain in the region is the only feature in Type-I and Type-II injuries.
b. Dislocation of the acromioclavicular joint with prominence of the lateral end of the clavicle is seen in Type-III injuries.



Figures 4.2A to D

(A) A normal joint with intact acromioclavicular and coracoclavicular ligaments; (B to D) Varying grades of injury classified as Type-I, Type-II, Type-III respectively. Acromioclavicular ligament—ACL; Coracoclavicular ligament—CCL.

Diagnosis

X-ray—A standard AP view of the shoulder may be deceptive at times. For better diagnosis, a stress view with the patient standing and holding a 5-kg-weight in the hand, with the hand by the side of the body, is useful. This demonstrates the subluxation/dislocation of the acromioclavicular joint.

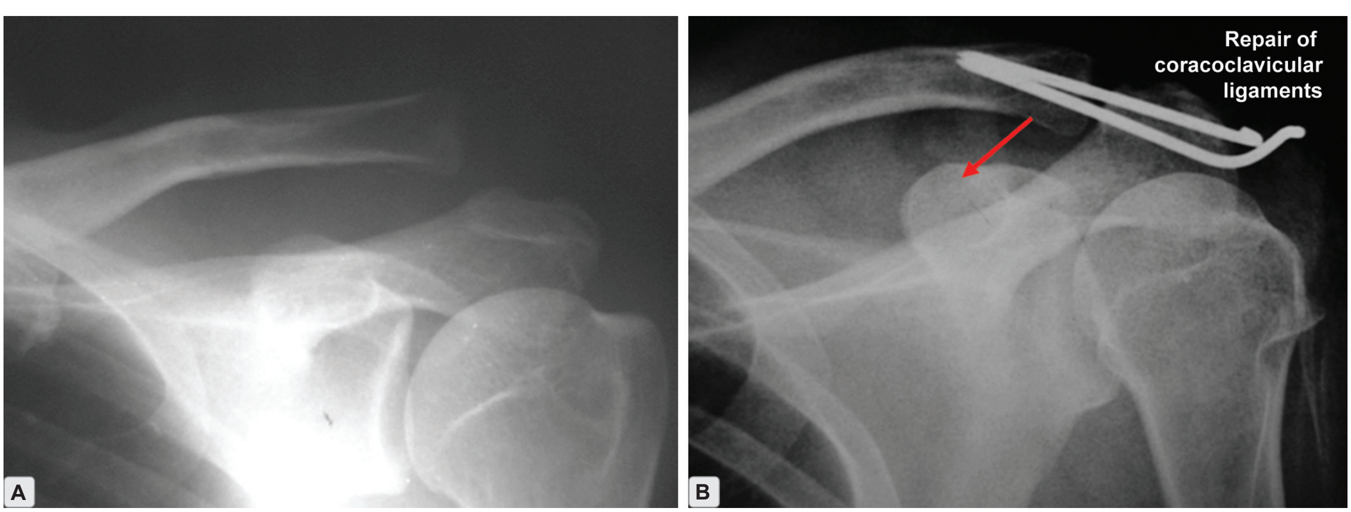
Management

Type-I and Type-II are treated nonoperatively in a shoulder arm sling. Type-III is treated surgically by the repair of the conoid and trapezoid part of the coracoclavicular ligament (Figs 4.3A and B). At the same time the joint is transfixed with 'K' wires.

In old unreduced acromioclavicular dislocation which is untreated, the projecting lateral end of the clavicle may be excised.

Revision Questions

Q. Discuss the anatomy of the acromioclavicular joint.
Q. What are the stabilizing structures of the acromioclavicular joint?
Q. Classify the acromioclavicular dislocation.
Q. Discuss the treatment of acromioclavicular dislocation.
Q. Discuss the complications of acromioclavicular joint dislocation.



Figures 4.3A and B

(A) Type-III acromioclavicular dislocation with disruption of both coracoclavicular and acromioclavicular ligaments; (B) After reduction, transfixation of acromioclavicular joint with two Kirschner wires and repair of the coracoclavicular ligaments as shown by the red arrow.

Essay Questions

Q. Discuss the pathoanatomy of the acromioclavicular joint.

Q. How do you classify the acromioclavicular joint dislocation? Outline the diagnosis and management.

DISLOCATION OF THE SHOULDER

When the head of the humerus loses its articulation with the glenoid cavity of the scapula, a dislocation of the shoulder occurs.

Note: *Dislocation is defined as a total loss of contact between the two articular surfaces.*

Types

a. Anterior—98%
b. Posterior—2%

Anterior Dislocation of the Shoulder

Mechanism: Occurs as a result of a hyperabduction and external rotation strain.

Types

a. Subcoracoid
b. Subglenoid
c. Subclavicular
d. Intrathoracic (rare) } associated with
e. Superior (very rare) } complications

Diagnosis

Signs and symptoms: Patient always presents with the injured limb held and supported by the help of other upper limb.

Following signs are seen:

a. Severe pain in the shoulder.
b. Loss of all the movements of the shoulder.
c. Loss of contour (prominence) of deltoid muscle bulge.
d. A hollow is appreciated at the shoulder.
e. Tenderness.
f. Head of the humerus is palpable in a place other than the glenoid.

Tests

The following tests are positive

a. **Duga's test:** Patient is asked to touch the opposite shoulder.

   If the patient is unable to touch the opposite shoulder, the test is positive.

   If the patient is able to touch the opposite shoulder, the test is negative.

   Note: *A negative test (Duga's) in the presence of anterior dislocation of the shoulder suggests that there is a fracture of the surgical neck of the humerus.*

b. **Hamilton ruler test:** When the head of the humerus is in the glenoid cavity, the bulging contour of the deltoid muscle prevents one from placing a ruler touching both acromion and lateral condyle of the humerus. In dislocation, this contour is lost and it is possible to place a ruler straight across the shoulder touching both the acromion and the lateral condyle of the humerus. The test is said to be positive.
c. **Bryant's sign:** It is positive when there is lowering and prominence of the anterior axillary fold.
d. **Callaway's test/sign:** Increase in girth of the shoulder.

Investigation: X-ray of the shoulder to know the type of dislocation and to ascertain the presence or absence of fracture. Commonest fracture associated with the anterior dislocation is fracture of the greater tuberosity of the humerus (Fig. 4.4).

Management: Anterior shoulder dislocation is always managed by closed reduction and immobilization in arm-chest bandage for a period of 3 weeks. Open reduction is indicated in late presentation (upto 3 weeks) and when the dislocation is irreducible by closed methods.

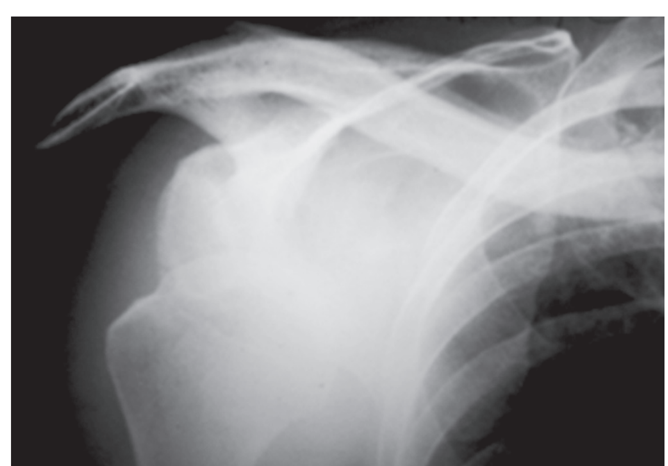


Figure 4.4
Subglenoid type of anterior dislocation of shoulder.

Note: *Open reduction can be safely tried within 3 weeks. Afterwards the procedure is associated with complications and should not be attempted. Injury to axillary artery, divisions of brachial plexus, redislocation and shoulder stiffness are some of the complications encountered.*

Left alone, the scapulothoracic rhythm gives considerable range of movement in an old non-reduced anterior dislocation of shoulder.

Method of Closed Reduction

a. *Kocher's method:* Patient under anesthesia, lies supine on the examination table. Traction is given with the elbow flexed and counter traction is given in the axilla with the help of folded towel by the assistant. Both traction and counter traction is given along the long axis of the humerus. The limb is slowly abducted and externally rotated. When the position which the head of the humerus had occupied just after the dislocation is attained, the dislocation gets reduced almost spontaneously because of the counter force. Once reduced then the shoulder is internally rotated, adducted and flexed and immobilized in arm chest position (Fig. 4.5A).
b. *Hippocrates method:* Patient under anesthesia, lies supine on the floor. The dislocated limb is held firmly by the surgeon at the wrist and traction is given. With the foot in the axilla the head of the humerus is gently maneuvered into the glenoid cavity (Fig. 4.5B).

Hippocrates method is a safer method when compared to Kocher's method. If not carefully executed, in Kocher's method, there is a possibility of a fracture of the surgical neck of the humerus occurring during the attempt. This is especially true if the patient is elderly and the bone is osteoporotic or when there is an occult fracture.

Recurrent Anterior Dislocation

If recurrent episodes of dislocation occur following the first episode, it is known as recurrent dislocation.

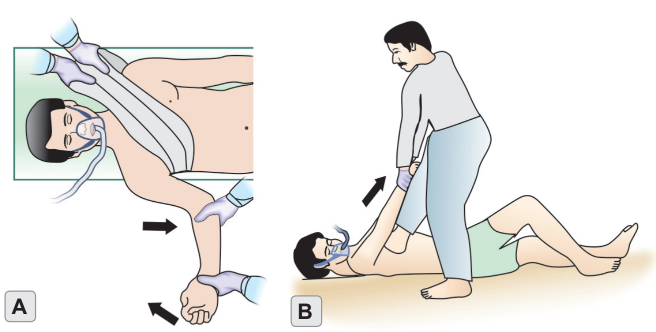
Pathology: Triple deformity (lesion) is responsible for such episodes. It includes:

a. *Bankart's lesion:* Tear of the anterior inferior glenoid labrum (2 o'clock to 6 o'clock in right shoulder: 6 o' clock to 10 o' clock in left shoulder).
   Diagnosis is established by MRI with contrast.
b. *Hill-Sach's lesion:* A defect in the posterolateral part of the head of the humerus.
   Diagnosis is established by a special view known as **Stryker notch view.** If in doubt, MRI has to be done for confirmation.
c. *A defect in the anterior inferior capsule.*

Stryker Notch View (Fig. 4.5C).

Patient is placed supine on X-ray table. The hand is placed on the head with the elbow pointing straight upwards. The cassette is kept underneath the shoulder.

The X-ray beam is directed cephalad with 10° inclination centering over the coracoid process. This picture gives an excellent view of Hill-Sach's lesion.



Figures 4.5A and B

(A) Kocher's method and (B) Hippocrates method of reduction of anterior dislocation of the shoulder. Note that in Hippocrates method the patient is on the floor. The foot of the surgeon is used as a lever and not to apply counter force.

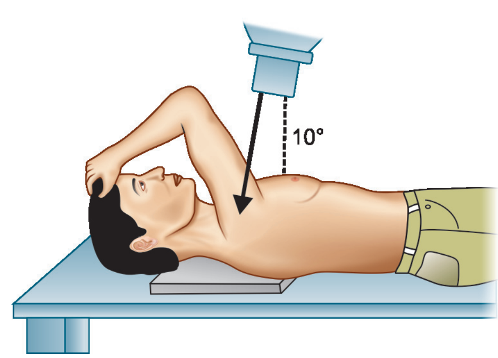


Figure 4.5C

Stryker notch view.

Management: Shoulder is stabilized surgically by selectively attending to the defects, e.g. Putti-Platt procedure, Bankart's procedure, Helfet–Bristow's procedure. These days stabilization procedures are done through shoulder arthroscopy too. (Refer for more details regarding arthroscopy).

Putti-Platt's Procedure

Described by Sir Harry Platt and Victoria Putti, this was a very successful procedure in the past. In this procedure, the subscapularis muscle was overlapped and shortened. The capsule of the shoulder joint was overlapped and tightened. The operation was based on the principle of double breasting of the muscle subscapularis and the joint capsule. This caused limitation of external rotation and prevented the defect from coming in contact with the glenoid during the movements of the shoulder. This open procedure is now rarely done.

Bankart's Procedure

In this operation, the anteriorly detached glenoid labrum is anchored back to the rim of the glenoid.

Original Bankart's operation was an open procedure. Today the same is done arthroscopically using suture anchors. Even bony Bankart's lesion can be managed by reattachment of the avulsed bony fragment to the glenoid arthroscopically.

Helfet Bristow's/Latarjet-Bristow's Procedure

This procedure was described by Helfet in the year 1958 and named after his mentor, Bristow.

The surgery involves osteotomy of the coracoid process along with the attached tendon of biceps and coracobrachialis and reattaching the same to the anterior inferior glenoid neck by making a horizontal slit in the subscapularis. This forms a strong dynamic buttress and acts as a block for the head of the humerus during abduction external rotation and prevents dislocation.

Helfet sutred the same to the neck of the scapula.

Latarjet described a similar procedure where in the osteotomized coracoid process is fixed to the neck of the scapula with a screw (Fig. 4.5D).

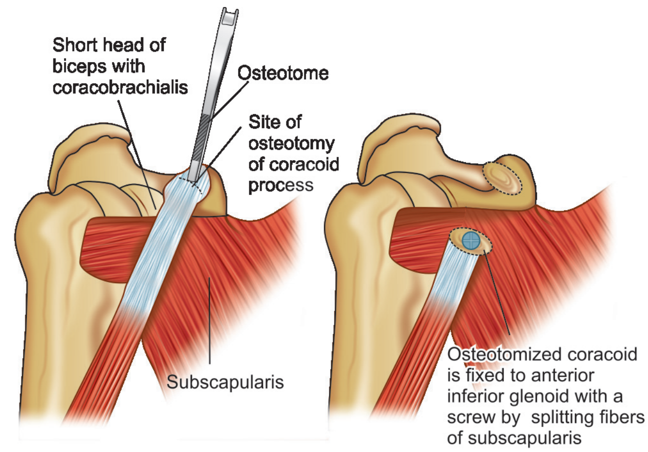


Figure 4.5D
Diagrammatic presentation of Latarjet-Bristow's procedure.

Posterior Dislocation of the Shoulder

Mechanism: A powerful adduction internal rotation strain, e.g. in seizures, electric shock or sometimes a direct anterior impact on the shoulder.

Types

a. Subacromial
b. Subglenoid
c. Supraspinous

Diagnosis

Symptoms and signs:

a. History of sustaining electric shock or an episode of seizures followed by severe pain and inability to move the shoulder.
b. Prominence of the coracoid process.
c. Limb is in a position of adduction and internal rotation.
d. Attempted abduction and external rotation is painful.

Investigation: X-rays—A standard AP view is taken. This gives an oblique profile of the glenoid. Following signs are seen:

a. *Loss of elliptical overlap:* Normally there is an overlap of at least one-third of the head over the posterior glenoid (obliquely profiled glenoid). This overlap becomes less or absent (Fig. 4.6).
b. *Empty (vacant) glenoid sign:* The glenoid cavity is void of articulating head of the humerus (Fig. 4.6).

Management: Reduction is achieved under anesthesia by giving gentle longitudinal as well as lateral traction followed by external rotation and abduction. Limb is

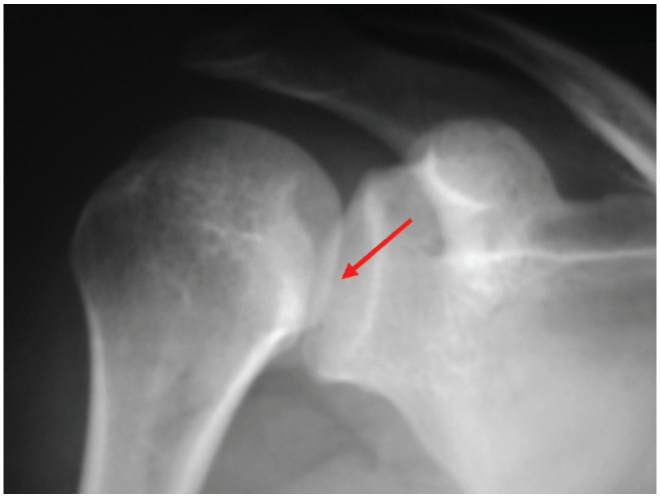


Figure 4.6
Loss of elliptical overlap as well as 'Empty glenoid sign' suggestive of posterior dislocation of the shoulder. This patient was a known epileptic and sustained this dislocation in an episode of seizure.

immobilized in external rotation and extension for a period of 3–4 weeks.

Recurrent Posterior Dislocation

It occurs as a result of posterior lesions.

a. Reverse Bankart's lesion of the glenoid.
b. Reverse Hill-Sach's lesion of the humerus.
c. Posterior glenoid deficiency.
d. Increased retroversion of the head and the glenoid.

Management: By surgical stabilization procedures, e.g. McLaughlin's procedure (Refer for details).

Luxatio erecta—It is an exaggerated form of infraglenoid type of dislocation of the shoulder. Patient presents with raised upper limb along with an inability to bring it down.

Revision Questions

Q. What are the different types of shoulder dislocation?
Q. Discuss the mechanism of shoulder dislocations.
Q. What are the clinical signs of anterior dislocation of the shoulder?
Q. Duga's test.
Q. Empty glenoid sign.
Q. What are the lesions responsible for recurrent anterior dislocation of the shoulder?
Q. Bankart's lesion.
Q. Hill-Sach's lesion.

Essay Questions

Q. Discuss the pathoanatomy, mechanism of injury, diagnosis and management of anterior dislocation of the shoulder.
Q. Define recurrent dislocation of the shoulder joint. Discuss the pathomechanics and management of recurrent anterior dislocation of the shoulder.
Q. Discuss the clinical features diagnosis and management of posterior dislocation of the shoulder. Enumerate the causes of recurrence.

DISLOCATION OF THE ELBOW

The elbow joint is a hinge joint which is inherently stable because of its design. It does not depend much on ligaments and other soft tissue structures surrounding it for its stability. Hence, considerable force is necessary to dislocate an elbow joint. Thus, in about one-third of the cases of elbow dislocation, fractures are commonly seen. Without fractures, the elbow dislocation is termed as simple and with associated fractures, it is termed as a complex dislocation.

Types

a. Posterior—90% (Figs 4.8A and B)
b. Anterior—10% (Fig. 4.7B)

Note: *Figure 4.7A shows an old unreduced posterior dislocation.*

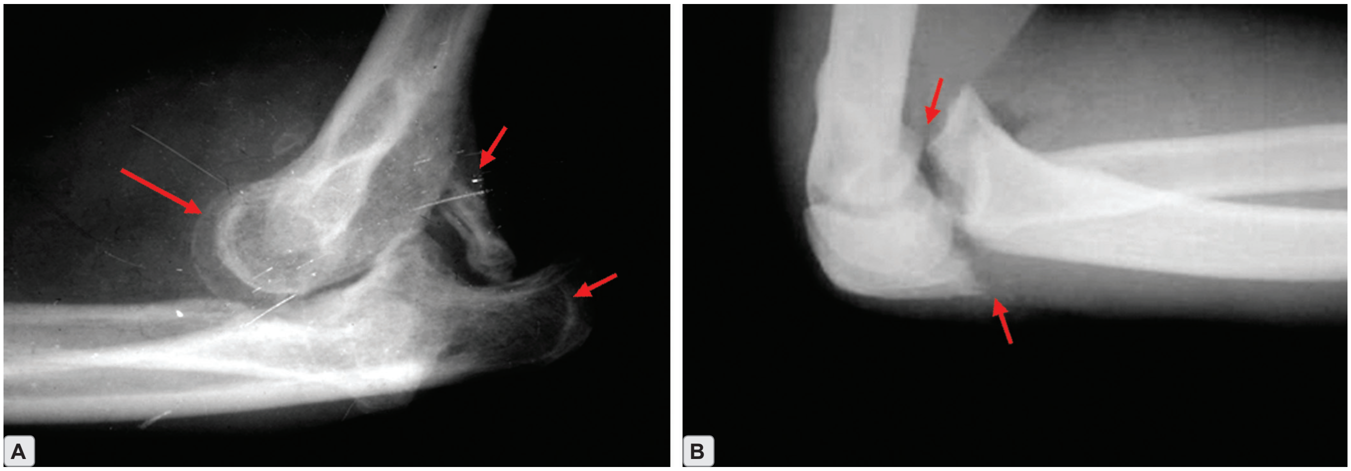
Mechanism of Injury

Posterior dislocation: Fall on an out-stretched hand with arm in abduction and extension, e.g. falling backwards after slipping (Most common fractures associated is the fracture of the medial epicondyle and radial head).

Anterior dislocation: A powerful blow to the posterior aspect of the elbow (Injuries of median nerve and brachial artery are common complications. Most common fracture associated is the fracture of the olecranon process.)

Diagnosis

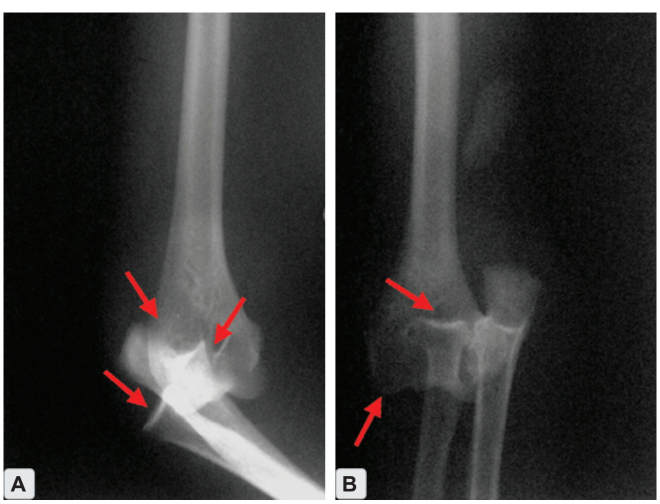
The posterior dislocation is diagnosed by clinical examination which shows loss of triangular relationship



Figures 4.7A and B

(A) An old unreduced posterior dislocation of the elbow with medial epicondylar fragment in the joint. In this scenario the elbow becomes irreducible unless the fragment is dislodged. (B) Fracture of the olecranon process of the ulna with anterior dislocation of the elbow. Carefully note the loss of articulation between the head of the radius and the capitulum, the coronoid process of the ulna and the coronoid fossa of the humerus as well as *in situ* position of the olecranon, which is articulating with the humerus in the flexed position of the elbow.

Also, there is no superior radioulnar joint disruption. These features suggest that there has been a direct blow to the elbow from behind causing anterior dislocation. The fracture of the olecranon process in this case is not an avulsion fracture. Generally fracture of olecranon occurs because of the pull of triceps.



Figures 4.8A and B

Lateral and anteroposterior view of the elbow showing traumatic posterior dislocation of the elbow joint. The displacement is posterior and medial.

of three bony points at the elbow and severe restriction of movements. Also, there is shortening of the forearm component of the limb. The arm component is normal.

X-ray proves the diagnosis conclusively and also determines the presence of fractures.

Management

a. Simple dislocations are managed by closed manipulation under anesthesia, reduction and immobilization for a period of 3 weeks. This is followed by gradual mobilization. An above elbow slab or cylinder cast is used for immobilization.
   Posterior dislocations are immobilized in flexion.
   Anterior dislocations are immobilized in extension.
b. Complex dislocations are managed by open reduction and stabilization.
   Associated fractures are anatomically reduced and fixed whenever possible. Badly comminuted radial head fracture requires excision at a later date.

Revision Questions

Q. Discuss the clinical diagnosis of posterior dislocation of the elbow joint.
Q. Classify dislocations of the elbow.
Q. Discuss the mechanism of injury of different elbow dislocations.

Essay Question

Q. Discuss the mechanism of injury, clinical features, diagnosis and management of different elbow dislocations.

DISLOCATION OF THE HIP JOINT

Hip joint is a ball and socket joint in which the spherical head of the femur articulates with the cup, i.e. the acetabulum. Inherent stability is imparted through this articulation. Strong ligaments, the tough capsule along with powerful muscles which surround the joint, impart additional stability. Among the ligaments the Y ligament of Bigelow, i.e. the iliofemoral ligament and the Ligamentum Capitis Femoris, i.e. the ligament of the head of the femur are the important stabilizers. Among the muscles, the Gluteus medius and Iliopsoas are the important stabilizers. Considerable force is necessary for a hip dislocation to occur (Trauma of severe magnitude).

Bigelow's Ligament—other names are Ligamentum Iliofemorale, 'Y' ligament, Bertin's ligament and Hipsiloid ligament. It is a Y shaped ligament with the apex attached to the anterior inferior iliac spine and the base diverged into medial and lateral bands, getting inserted to the intertrochanteric line. This imparts a 'Y' shape to the ligament. The medial band is attached to the lower aspect of the intertrochanteric line and the lateral band is attached to the tubercle at the upper aspect (Fig. 4.9).

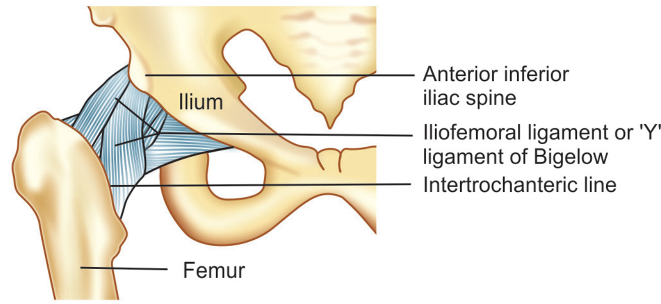


Figure 4.9

Ligaments supporting the hip joint.

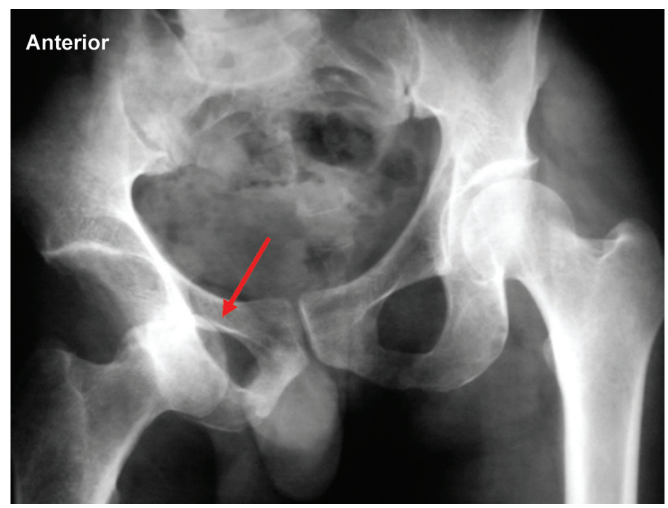


Figure 4.10

Obturator type of anterior dislocation of the hip. It is known as obturator type because the displaced femoral head is in the region of obturator foramen.

Types

The basic types of hip dislocation are:

a. Anterior:
   - Obturator (Fig. 4.10)
   - Pubic
   - Perineal
b. Posterior:
   - Gluteal
   - Sciatic
   - Ilial
c. Central (Fracture dislocation).

Mechanism of Injury

Anterior Dislocation

Hyperabduction and external rotation strain causes anterior dislocation of the hip joint. In olden days when sea voyage was common, the classical mechanism was that of having one leg on the boat and the other leg on the shore and the boat moved. The leg on the shore (the one which is more fixed) used to dislocate. These days skidding of a speeding two wheeler imparts a similar stress. Also seen when there is a landslide occurring in a mine, wherein a miner is stooping forwards and working, on whose back a heavy mass of mud falls. Under such condition, the hip goes into hyperabduction and external rotation and dislocates anteriorly (Fig. 4.11).

The head of the femur occupies a place below the acetabulum, either in the obturator, pubic or perineal region.

Posterior Dislocation

Classical mechanism is a 'Dash board injury'. In a road traffic accident, when there is 'Head on' collision, the dash board of the vehicle hits the knee. The position of the lower limb at the time of impact is that of flexion, adduction and internal rotation. Thus, an anterior impact of the crashing dash board on to the knee pushes the

head of the femur out of the acetabulum in a posterior direction (Figs 4.12A and 4.13).

The head of the femur occupies a place above the acetabulum, either in the gluteal sciatic or ilial region.

Central Dislocation

The classical mechanism is an impact from the side over the trochanter of the femur in an adducted limb (Fig. 4.12B).

Figure 4.11

Mechanism of injury for an anterior dislocation of hip joint which is shown here giving an example of a miner getting injured in a land slide.

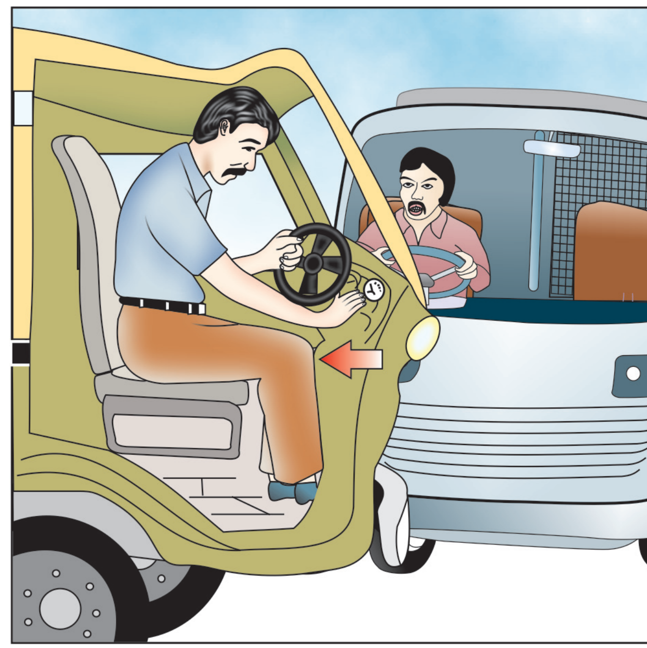
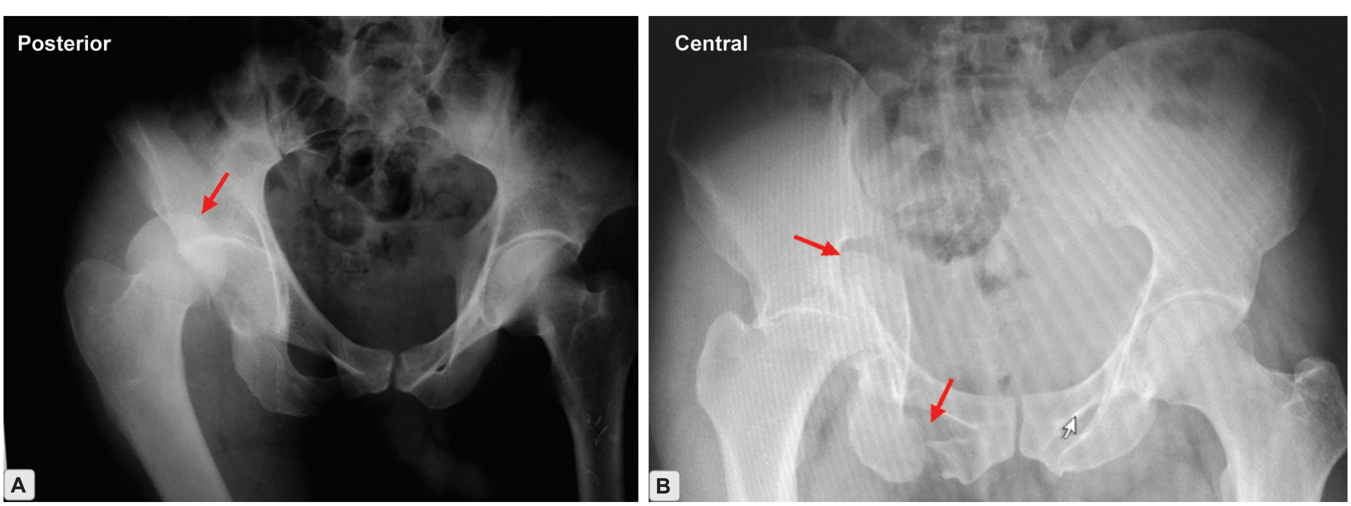


Figure 4.13

Mechanism of injury for a posterior dislocation of hip joint. The picture shows the classical impact of a dashboard in a 'Head on' collision.



Figures 4.12A and B

(A) Anteroposterior view showing traumatic posterior dislocation in a young patient. The femoral head is in the region of the sciatic notch. Hence, it is known as sciatic type (B) Pelvis with both hips showing fracture of the pelvis with central dislocation of the hip. Note that the distance between the symphysis pubis and the greater trochanter is decreased when compared to the opposite side.

Classification of Posterior Dislocation

Thompson-Epstein Classification

This classification system is based on radiographic findings. Based on this classification, the posterior dislocation of the hip is classified into five types.

- Type I: Dislocation with or without minor fracture.
- Type II: Dislocation with a single, large, fracture of posterior acetabular rim.
- Type III: Dislocation with comminuted fracture of rim of acetabulum, with or without major fragments.
- Type IV: Dislocation with fracture of the floor of acetabulum.
- Type V: Dislocation with femoral head fracture.

Stewart and Milford Classification

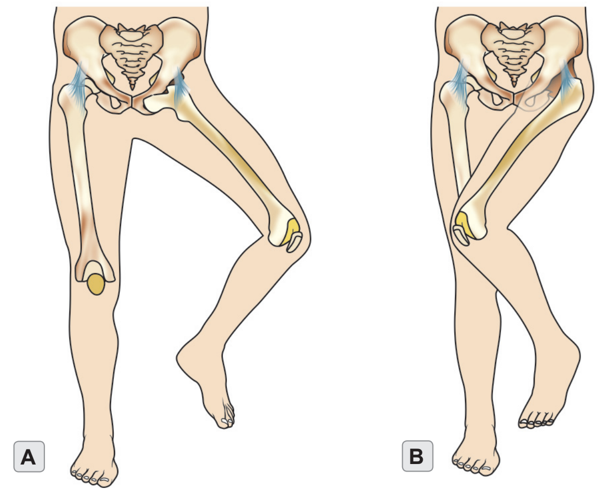
This classification is based on functional hip stability.

- Type I: Dislocation with no fracture or an insignificant fracture.
- Type II: Dislocation with a single or comminuted posterior wall fracture, but a functionally stable hip.
- Type III: Associated with gross instability of the hip joint secondary to loss of structural support.
- Type IV: Associated with femoral head fracture.

Diagnosis

- Diagnosis is made by clinical signs and X-ray.
- Classical clinical attitude of the lower limb in various types of dislocation is as follows:
  - Anterior dislocation: Flexion, abduction and external rotation with apparent lengthening (Fig. 4.14A).
  - Posterior dislocation: Flexion, adduction and internal rotation with apparent shortening (Fig. 4.14B).
  - Central dislocation: Adduction and neutral/ external rotation with signs of fracture pelvis. Shortening is mimimum.
- In addition to these classical attitudes, there will be severe painful restriction of all the movements of the hip joint.
- Radiograph confirms the diagnosis. The classical attitude of the limb is also made out in the AP view. Hence there is no need to take a lateral view unless an irregular dislocation is suspected (See Note: below).

Computed tomography (CT) and Magnetic resonance imaging (MRI) are recommended when there is fracture of the acetabulum or the femoral head for detailed evaluation and planning of appropriate treatment. They give valuable information about the shape and vascularity of the head of the femur and the acetabulum.



Figures 4.14A and B

The classical attitudes in anterior and posterior dislocation of the hip joint. (A) Anterior dislocation; (B) Posterior dislocation (refer text).

Management

Most of the simple dislocations without fracture are reducible by closed methods except when the head is entrapped in the torn capsule or muscle mass. All the closed maneuvers are done under general anesthesia. If a force in one direction in a particular position of the limb has driven the head of the femur out of the socket (acetabulum), a similar force in opposite direction should bring it back to the socket. Thus, the basic principle of reduction is to provide a counter force after achieving that position of the limb which was present immediately after the dislocation. Following reduction the limb is immobilized in a Thomas' splint for a period of 3 weeks in order to achieve good soft tissue healing.

Anterior Dislocation

Allis' method: The position of the patient is supine. The limb is held in flexion, abduction, external rotation at the hip (dislocated position) and flexion at the knee. Traction is given by the surgeon along the long axis of the femur while the assistant gives countertraction at the pelvis. Next, a second assistant gives lateral traction to the thigh (femur) and the surgeon slowly internally rotates and adducts the limb. Finally, when the dislocation gets reduced the limb (Fig. 4.15) is extended.

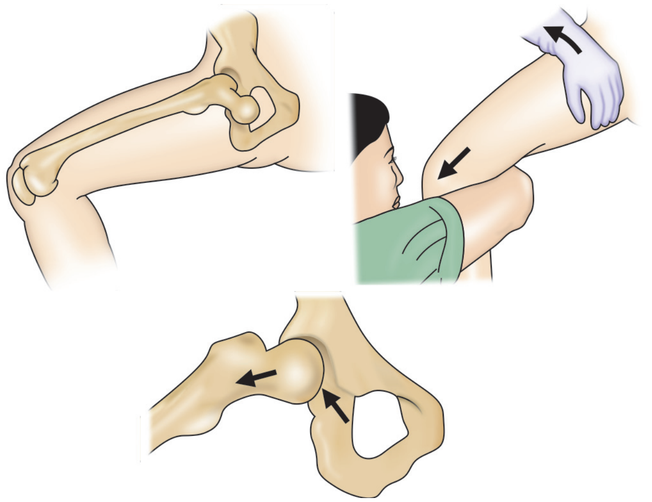


Figure 4.15
Allis' method of reducing the anterior dislocation of the hip. The arrows show the direction of the pull.

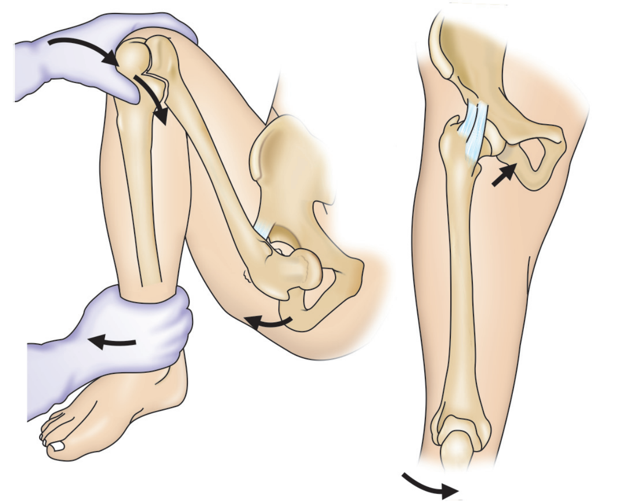


Figure 4.16
Reverse Bigelow's method for reduction of anterior dislocation of the hip.

Reverse Bigelow's Method

In this method, circumductory movement is given in an inward direction at the hip. Note that this is exactly opposite to Bigelow's method of reducing a posterior dislocation of the hip.

With patient supine the assistant stabilizes the pelvis and the surgeon does the maneuver beginning from the same flexed abducted and externally rotated position of the limb (Fig. 4.16) with the knee in flexion.

Posterior Dislocation

Allis' method: With the patient is supine and the limb in dislocated position of flexion, adduction, internal rotation, longitudinal traction is given by the surgeon. The assistant stabilizes the pelvis (countertraction). Next with the traction force acting, the limb is gently abducted, externally rotated and finally when the dislocation gets reduced the limb is extended (Fig. 4.17).

Bigelow's method: Position of the patient supine. The pelvis is stabilized by the assistant. The surgeon does the maneuver as follows; with knee in flexion hip is gently flexed to 90° and rotated in a circumductory manner in an outward direction till reduction is achieved (Fig. 4.18).

Stimson's method: Considered to be the least traumatic as gravity is made use of in this method. The position of the patient is prone and the limb is hanging out of the table, with hip and the knee flexed to 90°. Traction

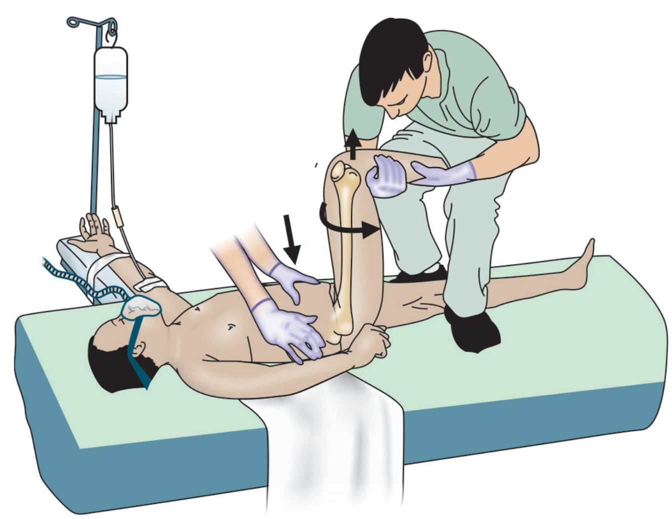


Figure 4.17
Alli's method of reduction for posterior dislocation of the hip joint.

force is applied just distal to the knee (over the upper third of the leg) by the assistant. The surgeon stabilizes the pelvis by applying pressure over the sacrum with the hand thus giving countertraction. Then with the other hand, the head of the femur is gently maneuvered into the acetabulum with the help of rotary movement of the limb given simultaneously by the assistant (Fig. 4.19) during maneuvering.

Note: *'Y' ligament of Bigelow acts as an anchor and helps in the reduction of dislocation. When the Y ligament of Bigelow is torn, the hip dislocation is known as irregular. Irregular dislocation is highly unstable.*

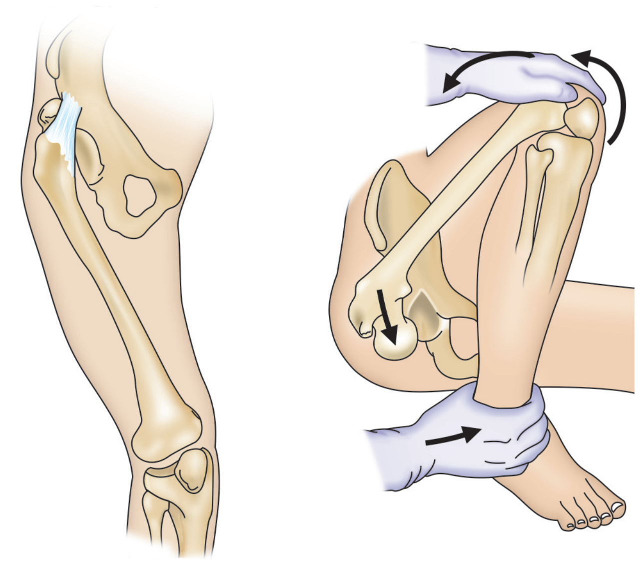


Figure 4.18

Bigelow's method of reduction for posterior dislocation of the hip joint.

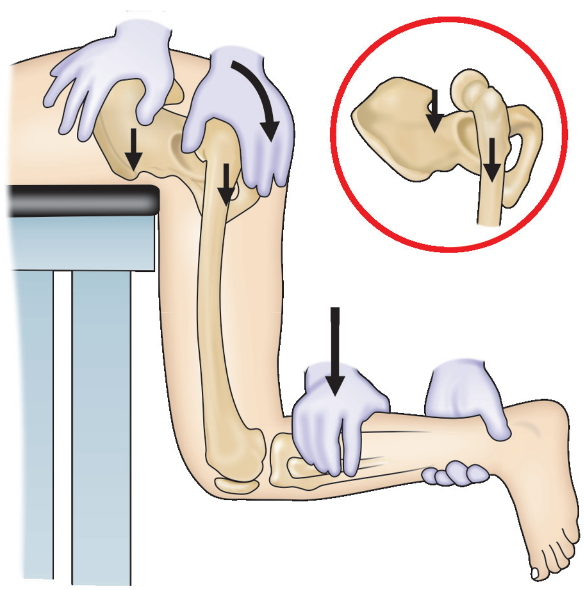
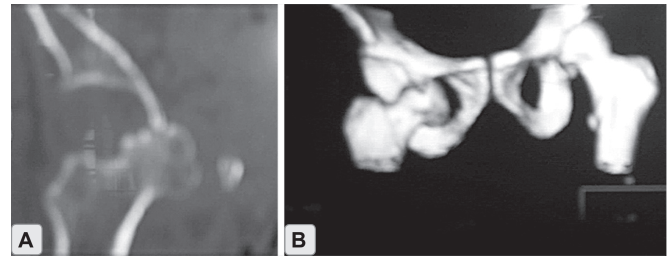


Figure 4.19

Stimson's method of reduction for posterior dislocation of the hip joint.

Complications

1. Irreducible dislocation (Figs 4.20A and B).
2. Avascular necrosis of the head of the femur and late degenerative arthritis (Figs 4.20A and B).
3. Sciatic nerve palsy (about 10%).
4. Associated fractures, e.g. fracture of the head, acetabulum, neck of the femur, etc.
5. Myositis ossificans (common after open reduction).
6. Recurrent dislocation (rare).



Figures 4.20A and B

The CT image of an old nonreduced anterior dislocation (4 months old) of the hip. Note the articular degeneration and the incongruity that has developed over 4 months which is not in favor of open reduction.

Revision Questions

Q. What are the basic types of dislocation?

Q. Write notes on:
   a. Pathological dislocation
   b. Paralytic dislocation
   c. Traumatic dislocation.

Q. What is Bigelow's ligament ? Discuss its role in hip dislocation.

Q. Discuss the pathoanatomy of the hip joint and classify hip dislocations. Explain in detail the different mechanisms responsible for hip dislocations.

Q. Discuss the different methods of reduction of posterior dislocation of the hip joint.

Q. Discuss the attitudes of the lower limb in different dislocations of the hip joint.

Essay Questions

Q. Discuss the mechanism of injury, diagnosis and management of posterior dislocation of the hip joint. Enumerate the complications.

Q. Discuss the mechanisms of injury, diagnosis and management of anterior dislocation of the hip joint.

Q. Discuss the role of CT scan in the evaluation of a hip dislocation. Discuss the management of a fracture dislocation of hip joint. Enumerate the complications.

Q. Discuss the mechanism of injury, the clinical features, diagnosis and management of a central fracture dislocation of hip joint.

DISLOCATION OF THE KNEE

Dislocation of the knee is considered to be a very serious injury. It results from a direct impact onto the

front of the knee by a force of considerable magnitude. The cruciate ligaments are torn and so are one or both of the collateral ligaments. Because of this the femur is pushed in a posterior direction over the tibia and a dislocation of the knee occurs.

Clinical Features

a. Bruising and contusion around the knee.
b. Presence of neurological and vascular deficit in the lower limb.
c. Signs of acute compartment syndrome may be present.

Diagnosis

X-ray gives a clear cut diagnosis. Always look for associated fractures, e.g tibial spine.

Treatment

It is an emergency. The dislocation has to be reduced as early as possible. Always a watch has to be kept on vascular status of the limb. Insufficiency can develop any time within a week. Doppler, pulse oximeter and when needed angiogram are of use in the diagnosis and treatment of vascular insufficiency.

Plaster immobilization is to be avoided. Also the knee should never be immobilized in extension. If stability is essential, external fixator application is a better option. Immediate ligament repair is not indicated in closed injuries. Ligament reconstruction is done at a later date. However, in open injuries repair of cruciate ligaments may be considered.

Essay Questions

Q. Discuss the mechanism of injury, diagnosis and management of a traumatic knee dislocation.
Q. What are the complications related to acute traumatic dislocation of the knee joint? Discuss their management.

Note: *Management of vascular injury and compartment syndrome has been discussed in Chapter 2 under Volkmann's ischemia.*

FURTHER READING

Shoulder Dislocation

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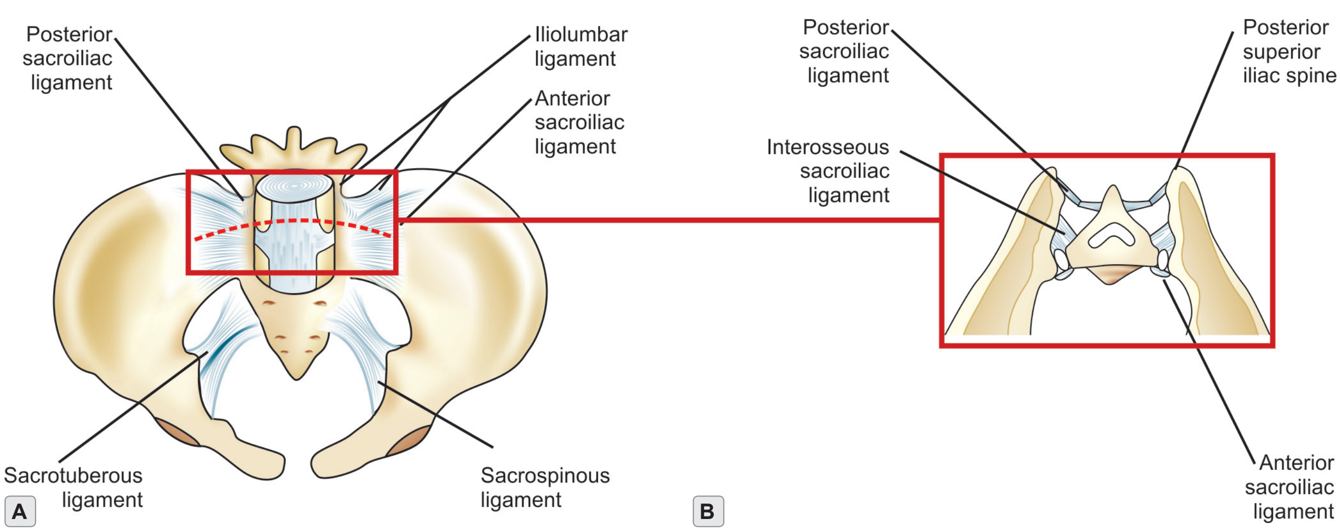
5 Fractures of the Pelvis

- *Structures imparting stability to the pelvis*
- *Deforming forces responsible for failure*
- *Classification*
- *Clinical features and investigations*
- *Treatment*

FORMATION OF PELVIS

The pelvis is formed by the sacrum articulating with a pair of hemipelvic bones formed by ilium, ischium and pubis, on either sides. Stability to the pelvis is imparted anteriorly by the pubic symphysis with its strong fibrocartilaginous cuff and ligament; posteriorly by the sacral articulation, with its strong ligaments, i.e. the anterior, interosseous and posterior sacroiliac ligament as well as the iliolumbar ligament. Additional stability is imparted by the muscles and soft tissues that are attached to the pelvis (e.g. sacrospinous and sacrotuberous ligament posteroinferiorly, gluteal muscles posteriorly, iliopsoas anteriorly) (Figs 5.1A and B).

Thus, it is an established fact that though the pelvis is the only link that connects the axial and the



Figures 5.1A and B

(A) The supporting ligaments that impart stability to the pelvis; (B) The cross-section of the inset showing the posterior stabilizers of the sacroiliac articulation.

appendicular skeleton, it has poor inherent stability and its stability entirely depends on integrity of soft tissues surrounding it and the strong ligament complex.

The pelvis has an inlet and an outlet allowing entry and exit of vital structures such as pelvic vessels and nerves, at the same time protecting them. It also has room for accommodating viscera.

For these reasons, the injuries of the pelvis have to be considered very seriously as there can be associated vascular, visceral and neurological compromise, leading to serious complications.

Further, the acetabulum may also get involved in pelvic fractures. Failure to evaluate and treat these injuries adequately may result in serious impairment of functions of the hip joint.

The pelvic girdle is formed by the 5th lumbar vertebra, the coxal bones (ilium, ischium and pubis), the sacrum and coccyx, bound firmly by capsule and ligamentous complex.

The pelvic ring is formed by the anterior arch and the posterior arch.

The anterior arch comprises of the pubic symphysis, the ilioischiopubic rami and the obturator foraminae.

The posterior arch comprises of the sacrum, the two iliac wings and the acetabulae.

Mechanisms of Injury (Figs 5.2A to C)

The injurious forces responsible are four in number.

a. External rotation force
b. Compression force
  - Anteroposterior
  - Lateral
c. Vertical shear
d. Combined

External Rotation Force

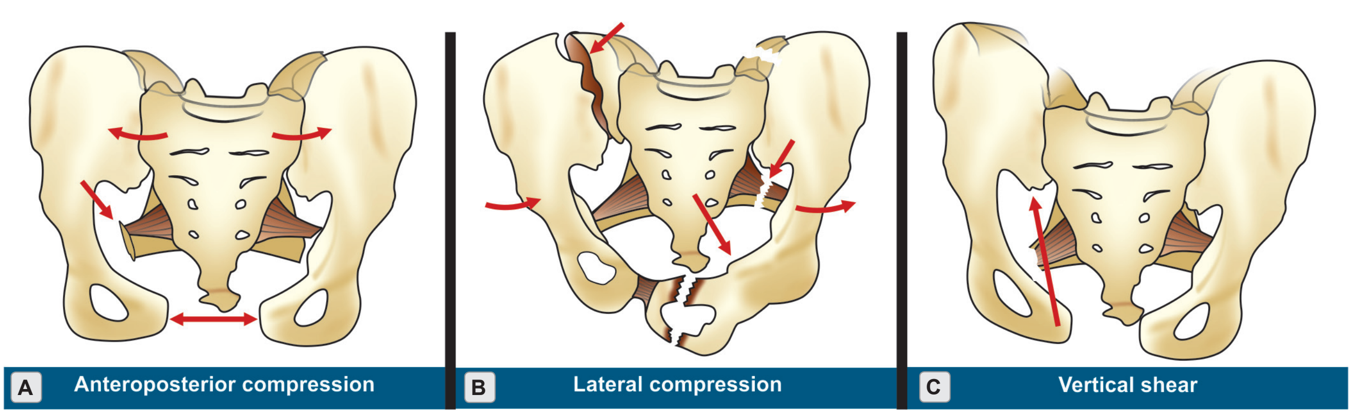
Forced external rotation stress on the lower limb causes disruption in an orderly manner from anterior to posterior which is described as follows.

Separation of symphysis pubis → opening of sacroiliac joint → stretching and disruption of sacrospinous ligament: ***Open book injury. Open book injuries are unstable in external rotation.***

Compression Force

Anteroposterior: (Analyzed taking into consideration posterior failure, i.e. sacroiliac joint, as the final event)

i. Without resistance from the opposite side e.g. direct vehicular impact.
   The failure starts anteriorly and proceeds posteriorly.
   The failure sequence is similar to open book injury but it occurs bilaterally.
   Results in very severe injuries. Visceral and vascular injuries are common.
ii. With resistance from the opposite side, e.g. caught between a wall and a vehicle.



Figures 5.2A to C
Nature of the fracture as per the direction of the force.

When there is resistance, anteroposterior compression occurring in the midline central plane results in a Butterfly like fracture fragment comprising of all the four rami anteriorly (bilateral superior and inferior pubic rami) and bilateral sacroiliac joint crushing/disruption, posteriorly.

Lateral:

i. Without resistance from the opposite side, e.g. direct vehicular impact.
   Anteriorly, results in closed book type of injury which manifests either as overlapping of symphysis pubis or ipsilateral/contralateral bucket handle bi-ramal fracture of the ilioischiopubic rami. Ipsilateral fracture of the ilium may also occur.
   Posteriorly, results in either crushing of the sacrum or disruption of interosseous and posterior sacroiliac ligament.
   These closed book injuries are unstable in internal rotation.
ii. With resistance from the opposite side, e.g. caught between a wall and a vehicle, fall of a tree or a heavy object over the pelvis in lying down posture.
   Very severe crushing occurs involving opposite hemipelvis. Visceral, vascular and neurological injuries are common.

Compression impact with resistance results in very severe crushing of the pelvis which is often not compatible with life because of associated vascular and visceral injuries. Hemorrhage is severe and may result in death even if timely medical aid is given. Morbidity rate is quite high. Mortality rate increases especially with open fracture pelvis. It rises to approximately 50%.

Vertical Shear

Results in vertical displacement of ipsilateral pelvic bone. ***These are unstable in both vertical and horizontal planes.*** Patterns of injury may be as follows:

i. Displacement occurring through disrupted symphysis pubis anteriorly and disrupted sacroiliac joint posteriorly (ligamentous disruption).
ii. Displacement occurring through disrupted symphysis pubis anteriorly and fractured ilium posteriorly.
iii. Displacement occurring through fractured ilioischiopubic rami anteriorly and disrupted sacroiliac joint posteriorly.

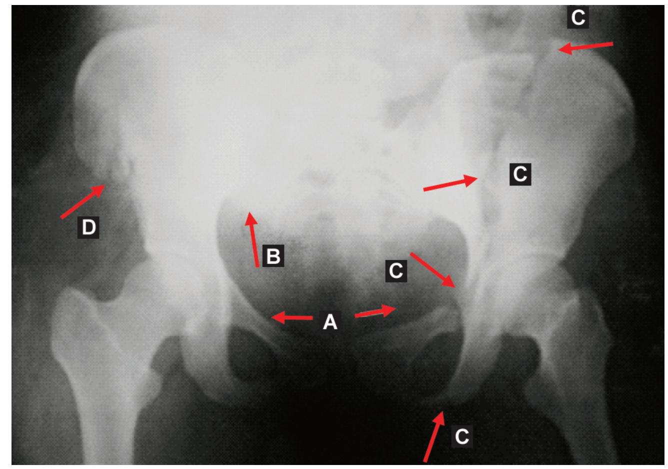


Figure 5.3

Fracture pelvis as a result of combined mechanism: (A) Diastasis of pubic symphysis; (B) Subluxation of (R) sacroiliac joint. Both the above injuries are as a consequence of 'open book' injury; (C) Vertical fracture extending through ischiopubic ramus and iliac wing (L). This is the result of vertical shear; (D) Avulsion fracture (R) ASIS*. This is the result of muscle pull. The fracture was an open fracture wherein more than one mechanism had operated.
*Anterior superior iliac spine—ASIS.

iv. Displacement occurring through fractured ilioischiopubic rami anteriorly and fractured ilium posteriorly.

Any of these four patterns may be seen depending on the direction of the force.

Combined

It is the result of mutiplanar stress causing ***high degree of instability*** (total instability). Shear, rotation, compression, translation, etc. more than one mechanism act together or in succession (Fig. 5.3). These injuries are generally fatal.

Classifications

Based on Tile's Classification (1995) as Follows

A. Rotationally and vertically stable fractures. These are subdivided into:
   A1. Avulsion fractures that do not interrupt the pelvic ring (Fig. 5.4).
   A2. Stable iliac wing fractures or minimally displaced pelvic ring fractures with minimal instability.
   A3. Transverse fractures involving sacrum and coccyx.

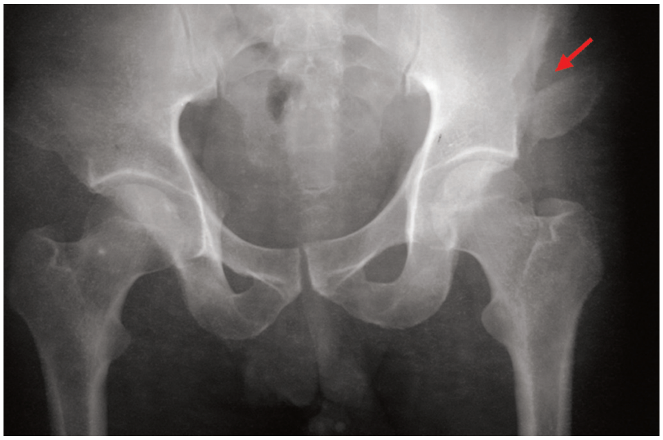


Figure 5.4

Tile's A1 fracture.

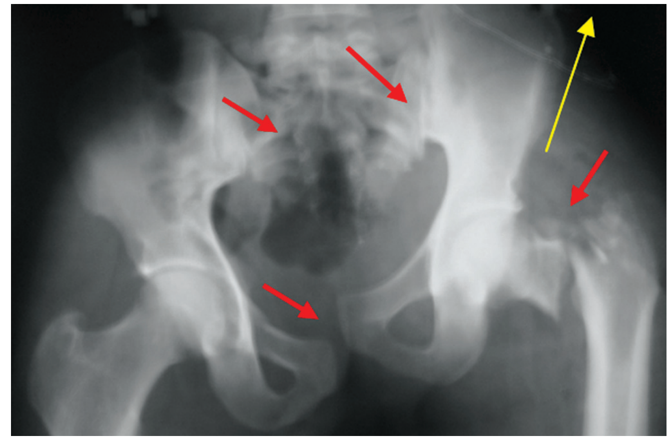
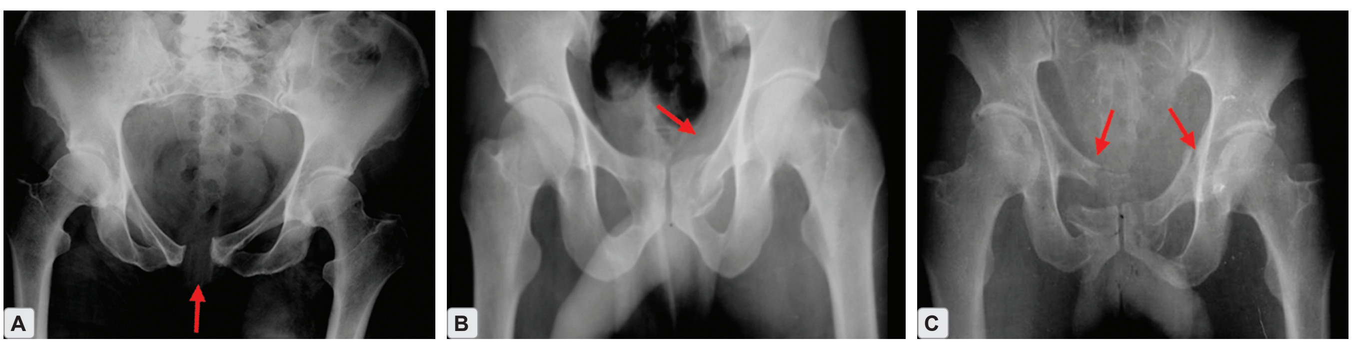


Figure 5.6

Tile's C3 fracture. Note the presence of sacral fracture on the opposite side which classifies this as type C3. Associated comminuted intertrochanteric fracture was an open fracture.



Figures 5.5A to C

Tile's B1, B2, B3 fracture.

B. Rotationally unstable, vertically stable fractures. Subdivided into (Figs 5.5A to C):
   - B1. Open book (due to anteroposterior compression).
   - B2. Closed book (due to lateral compression).
   - B3. Bilateral type B injuries.

C. Rotationally and vertically unstable. Subdivided into:
   - C1. Unilateral.
   - C2. Bilateral with one side type C and contralateral side type B.
   - C3. Bilateral with both sides type C (Fig. 5.6).

Sacral Fracture Classification

Based on Denis' classification, these fractures are classified according to their zone of injury.

Zone I Involvement of alar region of sacrum. (Neurologic complication least 6% approx)

Zone II Involvement of sacral foramina. (Neurologic complication moderate 30% approx)

Zone III Involvement of central canal. (Neurologic complication rate 60% approx. Also known for bowel and bladder involvement and sexual dysfunction).

Clinical Signs of Importance

1. Destot's sign: A large hematoma seen in the inguinal region or the scrotum.
2. Roux sign: Distance between the greater trochanter and the pubis is decreased.
3. Earle sign: A tender bony prominence or a large hematoma felt during per rectal examination.
4. Bleeding through urethra, vagina and rectum indicate visceral complications.

Investigations

It is very important to assess a pelvic fracture both qualitatively and quantitatively. Hence, proper radiological views are absolutely essential to detect the disturbance in 'Radiographic U'.

a. Standard AP view
   - Identifies fractures.
b. Pelvic inlet view, discloses (Fig. 5.7A)
   - AP displacement of pelvis
   - Inward/outward rotation of iliac wing
   - Sacral impaction or alar fractures
   - Avulsion fracture of the ischial spine
c. Pelvic outlet view, discloses (Fig. 5.7B)
   - Superior displacement of pelvis
   - Sagittal plane rotation
   - True AP view of the sacrum
   - Avulsion fracture of L5 transverse process.

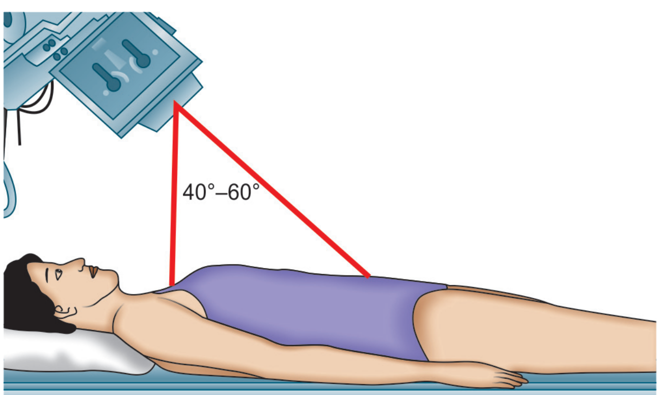


Figure 5.7A

Inlet view: The X-ray beam is directed 40°–60° caudal, centered at the level of anterior superior iliac spine.

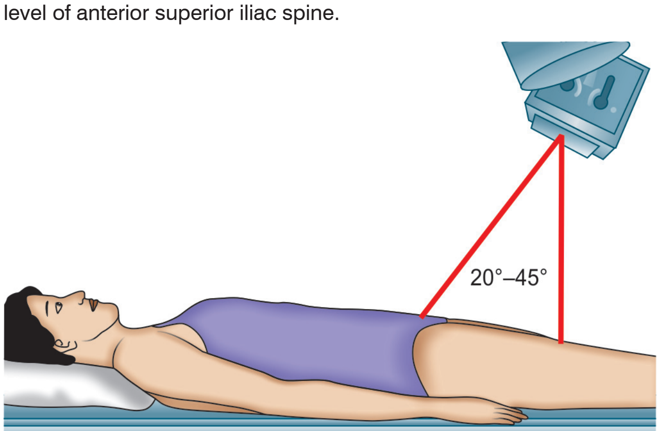


Figure 5.7B

Outlet view: The X-ray beam is directed 20°–45° cephalad and centered about 2 inches lower to symphysis pubis.

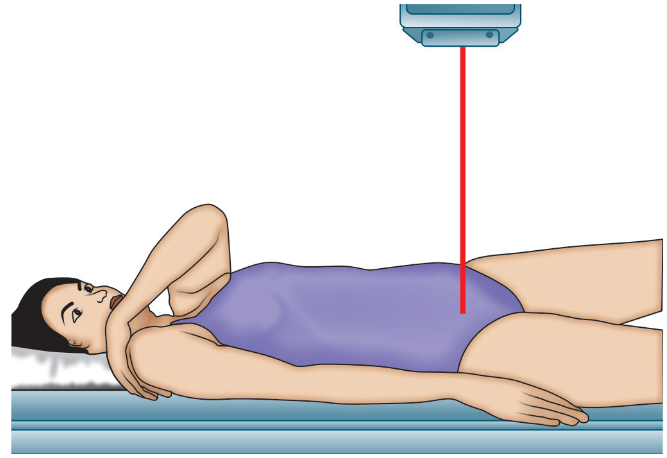


Figure 5.7C

Judet view: The patient is lying down in 45° oblique position. The beam is centered over the mid point of the pelvis.

d. Judet view/Oblique view (Fig. 5.7C)
   - Evaluates hemipelvis
     (The special views to be taken in pelvis fracture are shown in Figures 5.7A to C).

In complex fractures, CT scan and 3D reconstruction is necessary to plan accurate treatment.

Principles of Management

a. Immediate assessment of general condition of the patient and administration of resuscitative, life, saving measures when necessary.
b. Emergency management of shock and hemorrhage and stabilization of other associated fractures when present.
c. Carefully evaluate the mechanism of injury to know whether injury is stable or unstable.
d. Multidisciplinary approach when vascular, visceral and neurological injuries are associated.
e. Early reduction and stabilization of pelvic fractures to prevent further complications.

Wrapping firmly in bed sheet, Pneumatic Antishock Garments PASG, Military antishock trousers MAST, Therapeutic embolization are some of the measures undertaken to stop hemorrhage.

Uncomplicated stable injuries (not involving the pelvic ring) are managed nonoperatively. Skin and skeletal traction are employed to reduce and immobilize the fracture or maintain the position after reduction of the fracture under anesthesia.

Complicated and unstable injuries (involving and disrupting the pelvic ring) are stabilized surgically by open reduction and internal fixation using reconstruction plates and screws. External fixator frames for reducing and maintaining the reduction can also be employed.

Some of the indications for the surgical procedure:
a. Diastases of pubic symphysis greater than 2.5 cm.
b. Sacroiliac joint dislocations.
c. Displaced sacral fractures.
d. Posterior or vertical displacement of the hemipelvis (>1 cm).
e. Rotationally unstable pelvic ring injuries.
f. Sacral fractures in patients with unstable pelvic ring injuries.
g. Displaced sacral fractures with associated neurologic injury.

Some of the contraindications for the surgical procedure:
a. Patients who are unstable and critically ill with multiple associated injuries.
b. Badly contaminated open fractures with inadequate wound debridement.
c. Crush injuries.
d. Morel-Lavallee lesion (closed degloving).

Note: *Morel Lavallee lesion is diagnosed by fluctuation (fat necrosis) occurring under the skin of the involved area as a result of shearing of the subcutaneous tissue from the underlying fascia. It is associated with high rates of bacterial contamination. Hence, debridement and drainage of such lesions is a must before operative intervention.*

Complications of Fracture Pelvis

Early

a. Visceral, e.g. injury to bladder, bowel and urethra, other abdominal organs and chest.
b. Vascular e.g. internal iliac artery, superior gluteal artery.
c. Neurological e.g. sacral fractures.
d. Shock and hemorrhage.

Late

Malunion with disrupted pubic and sacroiliac articulation resulting in severe morbidity and impairment of physical function.

MALGAIGNE FRACTURE (FIG. 5.8)

It is a vertical shear fracture described by Malgaigne in the year 1885. It is highly unstable and characterized by anterior failure occurring through both the rami or through the symphysis pubis and posterior failure (with a massive disruption) occurring through the sacrum or the sacroiliac joint or the ilium consequent to severe trauma. Inadequate treatment results in significant late complications which are as follows:

Figure 5.8

A malunited Malgaigne fracture. Note the displacement as a result of vertical shear and malunion in the displaced position.

a. Paresthesias in the ipsilateral lower extremity.
b. Gait disturbances with significant limp.
c. Severe low back pain.
d. Groin pain.
e. Neurologic abnormality.
f. Leg length discrepancy.
g. Pelvic obliquity.

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6 Spinal Injuries

- *Structure and functions of the spinal column*
- *Mechanisms of injury and concepts*
- *Specific types of fractures and incomplete cord injury syndromes*
- *Management*
- *Signs of grave prognosis and a ray of hope*

Spine is a marvel of creation. It is a flexible structure having many functions, yet remains stable throughout and protects the vital structures within.

Structure of the Spinal Column

The basic unit of this structure is a vertebra. At birth there are 33 vertebrae; 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 coccygeal. In adults there are only 24 vertebrae because of the fusion occurring in the sacral and coccygeal region resulting in the formation of the bone sacrum and coccyx. The curvature of the spinal column also changes from straight at birth to an S-shaped one in adulthood.

Each vertebra consists of a body and a posterior arch. The body is made up of a block of spongy bone and between the bodies of the two vertebrae is the fibrocartilaginous intervertebral disc which binds the bodies of the adjacent vertebrae. The posterior arch consists of a spinous process, two transverse processes and a lamina. The arch is attached to the body by two pedicles. There are two articular facets on the posterior arch on each side, i.e. superior and inferior, which help in the articulation of the adjacent vertebrae, i.e. with the one above and the one below. Thus, the spinal column is formed. The central canal within the vertebra is bounded anteriorly by the body, laterally and posteriorly by the posterior arch. The spinal canal harbors the spinal cord.

Additional support and stability is given to the spinal column by the ligaments. They are from anterior to posterior as follows.

i. The anterior longitudinal ligament binding the vertebral body anteriorly
ii. The posterior longitudinal ligament binding the vertebral body posteriorly
iii. The ligamentum flavum binding the adjacent laminae
iv. The interspinous ligament extending between the spinous processes and binding them
v. The supraspinous ligament extending over the spinous processes and binding them.

Note: *The names themselves describe their location (Fig. 6.1) except ligamentum flavum.*

Structures that Impart Stability to the Spinal Column

Anterior Interbody Synarthrosis

Comprises of the intervertebral disc binding the adjacent bodies of the vertebra along with the anterior and posterior longitudinal ligaments.

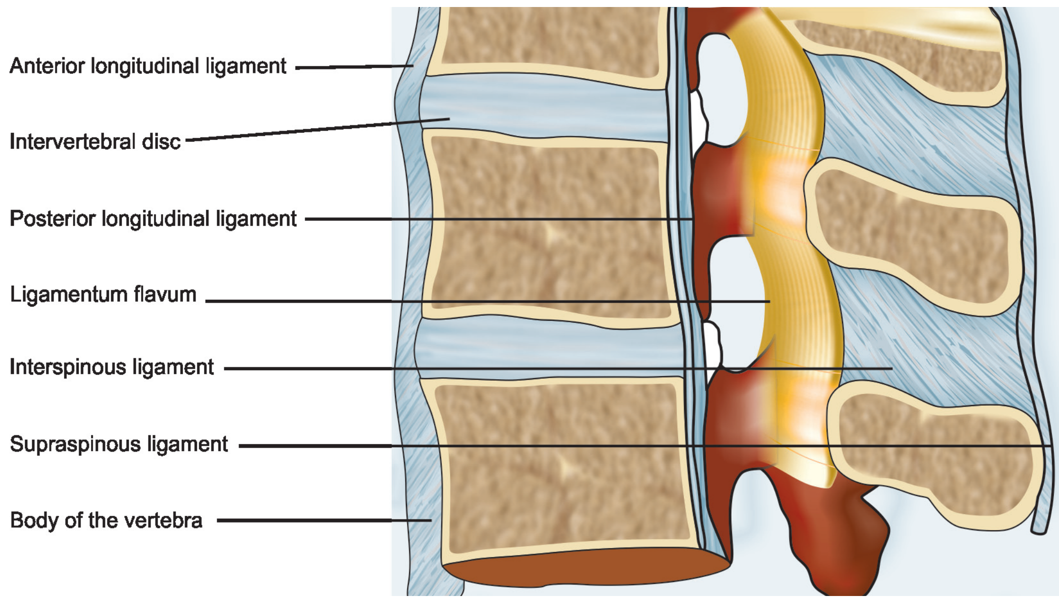


Figure 6.1

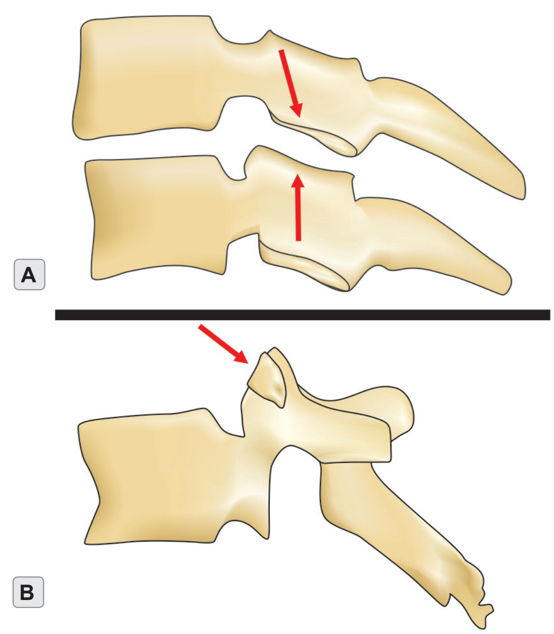
Ligamentous structures which impart stability to the spinal column.

Posterior Ligament Complex

The posterior ligament complex binds the posterior elements (posterior arch) and consists of the supraspinous ligament, the interspinous ligament and the ligamentum flavum.

The Facet Joints (Figs 6.2A and B)

The direction of the facet joint is almost transverse in the cervical spine, anteroposterior in the thoracic spine and mediolateral in the lumbar spine. Thus, the facetal articulation in the cervical spine is less stable when compared to thoracic and lumbar articulation.



Figures 6.2A and B

The direction of facet joint in (A) cervical and (B) thoracic vertebra.

Functions of the Spinal Column

1. Provides protection of the spinal cord and associated nerves.
2. Supports the body frame in an upright position.
3. Supports vital organs.
4. Provides a structural foundation for the shoulder girdle and the pelvic girdle.
5. Acts as a shock absorber on load-bearing.
6. Provides attachment to the thoracic cage.
7. Provides attachment to the muscles.
8. Allows several movements such as flexion, extension, rotation, etc.

Hence, spinal injury results in serious disability as it affects several functions.

SPINAL INJURY

The spinal injury has two components.

a. Injury to the spinal column, i.e. bony injury.
b. Injury to the spinal cord and nerve roots, i.e. neural injury.

It is possible to have a spinal column injury without spinal cord injury and vice versa, i.e. spinal cord injury without column injury OR both the injuries together, i.e. spinal column and spinal cord injury.

Denis' Three Column Concept (Fig. 6.3)

The spine has been divided into three columns namely, anterior, middle and posterior by Denis. He described that the failure of one or more columns occurs in spinal injury when deforming forces cause axial compression, axial distraction or translation.

Anterior column: Consists of anterior longitudinal ligament, anterior half of the vertebral body and the anterior portion of annulus fibrosus.

Middle column: Consists of posterior longitudinal ligament, posterior half of the vertebral body and the posterior portion of annulus fibrosus.

Posterior column: Consists of the posterior elements (neural arch) with ligamentum flavum, interspinous and supraspinous ligament, facet joints and the capsule.

Mechanisms of Spinal Injuries

Several mechanisms can disrupt the spinal column causing fracture, dislocation or fracture dislocation. Depending on the severity of injury and the compromise of stabilizing structures, the injuries are diagnosed as *stable* or *unstable (Nicoll's classification).*

Basically, these injury mechanisms are classified based on the deforming effect of an indirect impact, direct impact or a traction force on the spine.

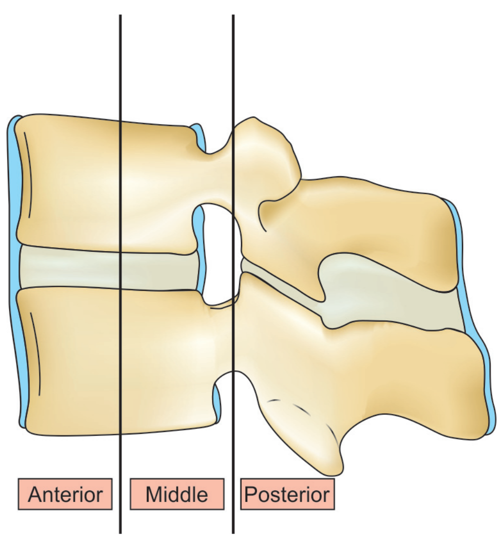


Figure 6.3

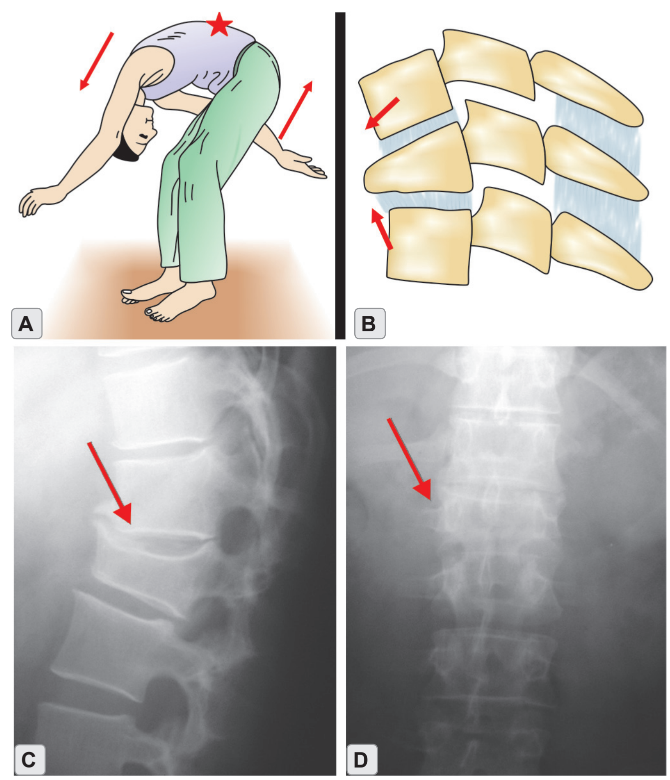
Denis' three column concept.

Indirect Mechanism

Injuries occurring as a result of this mechanism are classified as:

a. Flexion injury
b. Flexion rotation injury
c. Hyperextension injury
d. Vertical compression injury
e. Shearing
f. Flexion distraction

Flexion injury: In this, the deforming force is unidirectional. It exerts a compression stress (closing type of stress) on the anterior structures and results in a wedge compression fracture of the body of the vertebra. The posterior elements do not fail. The injury is diagnosed as unstable when there is an evidence of compression fracture which is more than 50% of height of the body. Otherwise, the injury is considered as stable (Figs 6.4A to D), e.g. fall from a height and landing on the feet. This event causes hyperflexion in the lower thoracic and upper lumbar region resulting in a wedge compression fracture.



Figures 6.4A to D

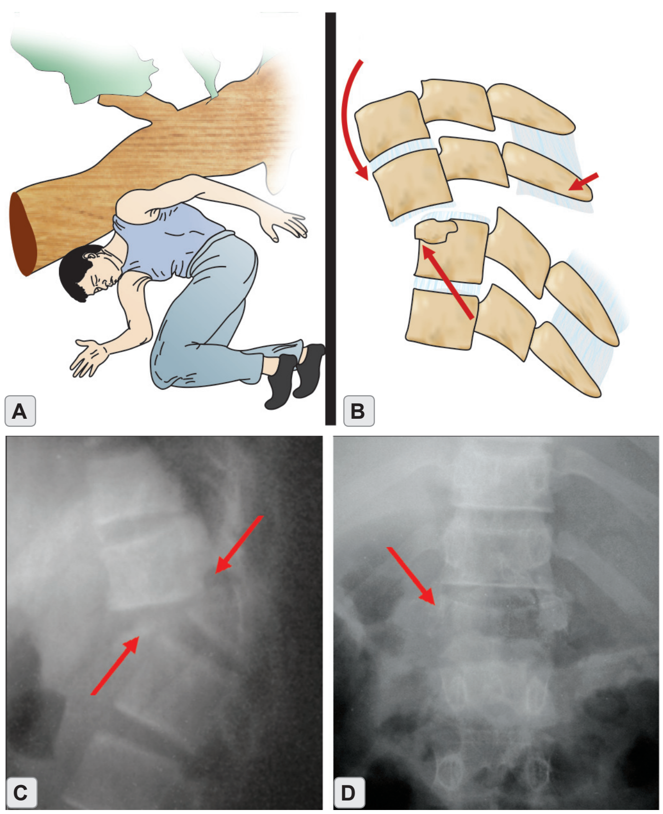
Pure flexion injury of the spine resulting in wedge compression of the vertebra. If the compression is less than 50% of the original height of the vertebra, the injury is considered as stable.

Flexion Rotation Injury

In this, the deforming force is multidirectional. Thus, in addition to causing fracture of the body of the vertebra and injury to the posterior elements (posterior ligament complex), the torsional stress in this mechanism disrupts the facet joint articulation also causing dislocation. This results in a fracture dislocation. Injury is highly unstable and probability of spinal cord injury is high (Figs 6.5A to D).

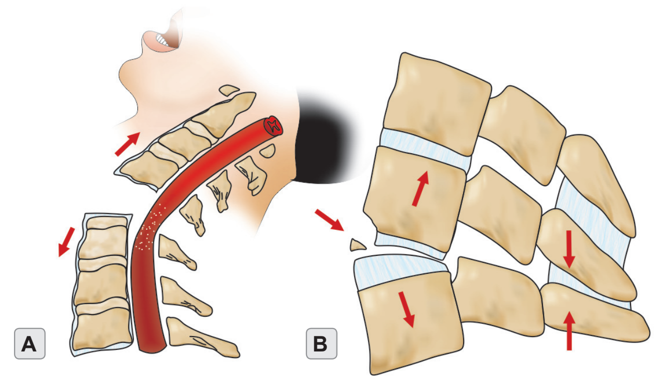
Hyperextension Injury

In this, the force is unidirectional. It exerts distraction stress on the anterior structures. Thus, the failure is mainly limited to anterior interbody synarthrosis. Sometimes, a small triangular fragment of bone is avulsed from the inferior vertebra due to the pull of the anterior longitudinal ligament. This feature is considered as a 'tell tale' evidence of a hyperextension injury (Figs 6.6A and B).



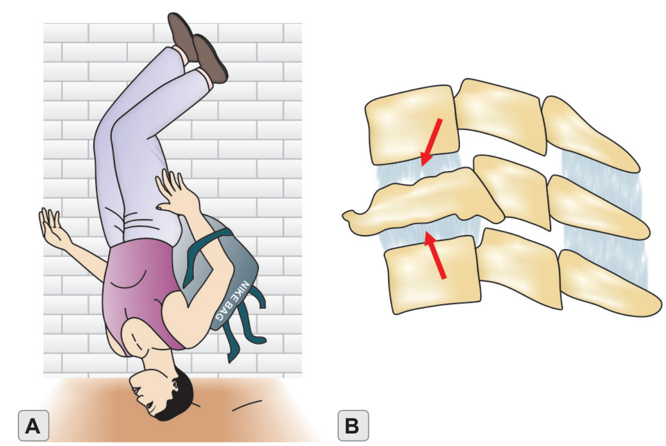
Figures 6.5A to D

Flexion rotation injury resulting in a fracture dislocation. The rotational element in the mechanism causes disruption of all the three stabilizing components namely, the anterior interbody synarthrosis, the facet joint articulation and the posterior ligament complex.



Figures 6.6A and B

Hyperextension injury. Note the direction of forces and site of distraction and compression. The small triangular fragment avulsed from the upper vertebral body when seen in the radiograph, indicates hyperextension injury.



Figures 6.7A and B

Burst fracture due to a vertical compression injury. The classical example is a fall from a height with a direct impact on the vertex/head OR a heavy object falling on the vertex.

Vertical Compression Injury

In this, the force is axially transmitted, the involved vertebra gets compressed axially between the two vertebrae and bursts into fragments. The disc material and the fragments of bone are retropulsed into the spinal canal. Burst fractures are unstable fractures and even if immediate neurological deficit is not observed, they tend to cause chronic instability and pain. The risk of ensuing neurological deficit always exists (Figs 6.7A and B).

Shearing Injury

In this, the force is transmitted transversely through the anterior interbody synarthrosis and posterior

ligament complex causing dislocation at the level of injury. Fractures of the pedicles and facet joints also are observed. The injury is highly unstable and spinal cord injury is common.

Flexion Distraction Injury

In this, the fulcrum of flexion is anterior to the body of the vertebra. The force may pass entirely through the bone, causing 'Chance fracture' OR through the posterior ligament complex.

'Chance fracture' (injury entirely through the bone) is stable after reduction. But, injury through the posterior ligament complex is unstable and needs surgical stabilization (Figs 6.9A and B).

Direct Mechanism

Direct penetrating injury, e.g. fire arms and knives, can lead to varying grades of spinal column and cord injury depending on the nature and potency of the injurious agent.

Traction Injuries

In this, the muscles and ligaments pull the bone and result in avulsion fracture, e.g. transverse process fracture, spinous process fracture, etc.

Specific Types of Spinal Column Injury

Jefferson's Fracture

Named after Goeffrey Jefferson, a British neurosurgeon who reported four cases in the year 1920.

It is a four part burst fracture of the C1 (the Atlas) vertebra involving the anterior and posterior arch as a result of axial compression, e.g. diving into a shallow pool, impact of the head against the roof of a vehicle, fall on the vertex, etc. Variants can occur as two or three part fractures.

The patient presents with pain in the neck. Neurological deficit is rare except when vertebral artery is involved which results in Wallenberg's syndrome (lateral medullary syndrome). Features suggesting the syndrome include, ipsilateral involvement of cranial nerves, Horner's syndrome, ataxia and loss of pain and temperature sensation.

Treatment depends on whether the injury is stable or unstable. This is evaluated by the amount of injury to the anterior arch and the intactness of the transverse

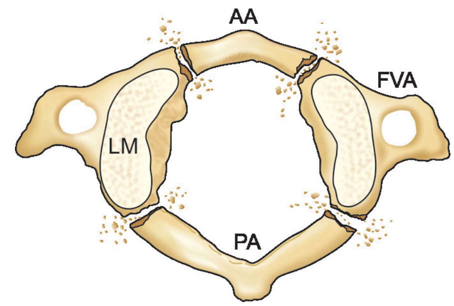


Figure 6.8

Jefferson's fracture. Anterior arch (AA); Posterior arch (PA); Lateral mass (LM); Foramen for vertebral artery (FVA).

ligament. Stable injury needs prolonged immobilization in a collar or halo traction (at least for 3 months). Unstable injury is treated by fusion of the first three cervical vertebrae (Fig. 6.8).

Chance Fracture (Lap Seat Belt Injury)

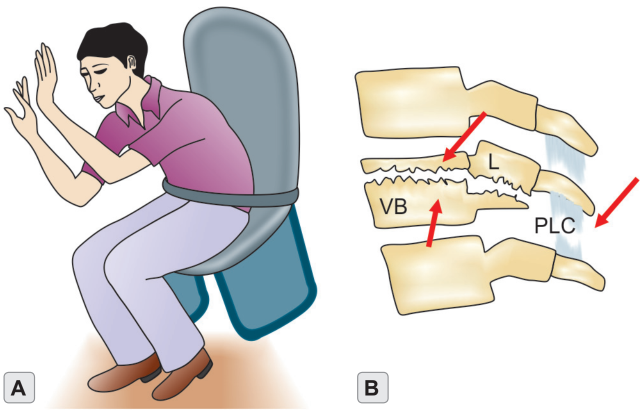
It was described by GQ Chance in 1948. It was common in motor vehicle accidents, when the vehicle was involved in a 'head on' collision and the passenger was wearing a lap seat belt. The incidence has sharply decreased since the implementation of shoulder restraint system in the seat belt. The injury occurs as a result of violent forward flexion. The posterior elements namely, the spinous process, lamina, the pedicle and the posterior part of the vertebral body fail as a result of distraction force resulting in characteristic transverse fracture whereas the anterior portion of the vertebral body fractures as a result of compression force. This injury, where all components are bony, is stable and heals uneventfully with immobilization.

But, if the posterior injury is that of failure of posterior ligament complex, even though the anterior injury is bony, the injury is unstable. So also, is pure ligamentous disruption. Incidence of cord involvement is high in these unstable injuries which occur as a variant of classical 'Chance fracture'. These unstable injuries need surgical stabilization (Figs 6.9A and B).

Hangman's Fracture

This fracture traditionally occurs after judicial hanging with the noose being placed below the chin. Hence, this name is given.

It is a fracture of both the pedicles OR the pars interarticularis of C2 (the Axis) vertebra as a result of



Figures 6.9A and B

'Chance fracture' (jack knife fracture) secondary to 'Lap seat belt' injury. Note that the injury shown has both bony and ligamentous component and hence is unstable (refer text). Vertebral body (VB); Lamina (L); Posterior ligament complex (PLC).

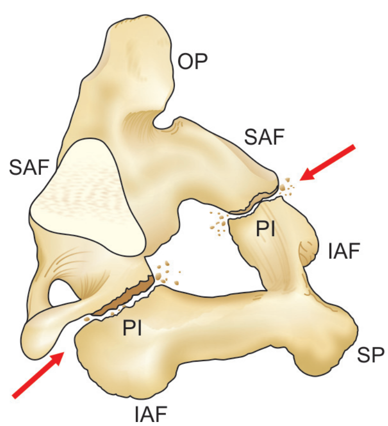


Figure 6.10

Hangman's fracture. Odontoid process (OP); Superior articular facet (SAF); Inferior articular facet (IAF); Pars interarticularis (PI) which is the portion between the two articular facets namely superior and inferior spinous process (SP)

distraction that occurs after the drop when the neck is forcibly hyperextended by the weight of the body. Similar mechanism operates in a road traffic accident when neck undergoes forcible hyperextension. The occiput strikes forcibly against the arch of the atlas which in turn forces itself into the pedicles of C2. This breaks the pars inter-articularis. The outcome of this injury, need not be fatal as in judicial hanging.

Minimally displaced fractures are treated by means of skeletal traction followed by hard collar/halo body jackect. Displaced fractures are treated by surgical stabilization and fusion (Fig. 6.10).

Clay Shoveller's Fracture

First seen among Australian Clay Shovellers who used to lift heavy loads of clay. The mechanism is an avulsion injury. As a result, avulsion fracture of the spinous process can occur from C6 to T1. Treatment is to immobilize the area with collar till good union is seen. Detailed evaluation for stability by taking flexion/extension radiographs, CT and MRI is necessary when the fracture involves multiple vertebrae, the laminae and the facetal joints. Such injuries should not be loosely branded as Clay Shoveller's fracture on the basis of spinous process fracture alone.

Whiplash Injury

Common in road traffic accidents when there is rear impact (rear-end collision). The neck is violently hyperextended and flexed. If the impact is less severe, it results in only soft tissue injury. If the impact is severe, it results in injury to intervertebral disc, the facet joints, ligaments, the muscles and the nerve roots. Concussion injury to the brain is also common. Depending on the severity, the patient may present with following symptoms.

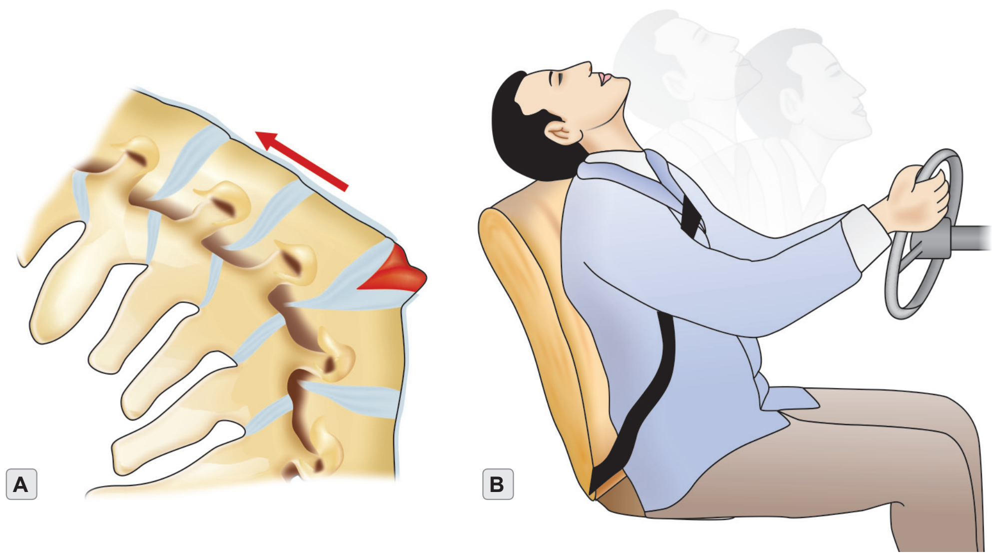
i. Pain and stiffness in the neck.
ii. Limitation of movements.
iii. Headache, dizziness, nausea, (result of concussion).
iv. Hoarseness of voice (involvement of recurrent laryngeal nerve).
v. Difficulty in swallowing (stretching and contusion of esophagus).
vi. Pain in the back and the shoulder (radiating pain).
vii. Pain and paresthesia in the limbs (due to involvement of nerves).

Symptoms may develop as early as within 2 hours of injury to as late as 8 days.

Radiographic evaluation is a must and in patients having symptoms of severe injury CT and MRI are indicated.

Soft tissue whiplash injury invariably heals in 2–3 weeks with immobilization in collar. Severe forms may cause mild degree of residual pain (Figs 6.11A and B).

Note: *Head rest support of the car seat considerably decreases the hyperextension and prevents Whiplash injury.*



Figures 6.11A and B
Mechanism of an hyperextension/whiplash injury.

Spinal Cord Injury

Spinal cord injury can be complete or incomplete. Complete transection of the cord results in Quadriplegia in cases of cervical spine injury and Paraplegia in cases of thoracic spine injury which is characterized by complete sensory loss and motor paralysis below the level of the lesion along with paralysis of urinary bladder and bowels.

Because the spinal cord ends at the lower border of L1 vertebra and only nerve roots occupy the canal below L1, any injury at or below the level of L1 will result in Conus medullaris and/or 'Cauda Equina' lesion.

For the same reason, in lesions between C1-L1, the level of the column injury does not correspond with the level of cord injury. Hence, a formula is derived to determine the level of cord injury.

In the cervical spine, add one to the level of the column injury, in the upper thoracic spine, add two and in the lower thoracic, add three to determine the level of cord injury. For example, if vertebral injury is at C4, the neurological level of injury is at C5; if the vertebral injury is at T4, the neurological level of injury is at T6; if the vertebral injury is at T12, the neurological level of injury is at L3.

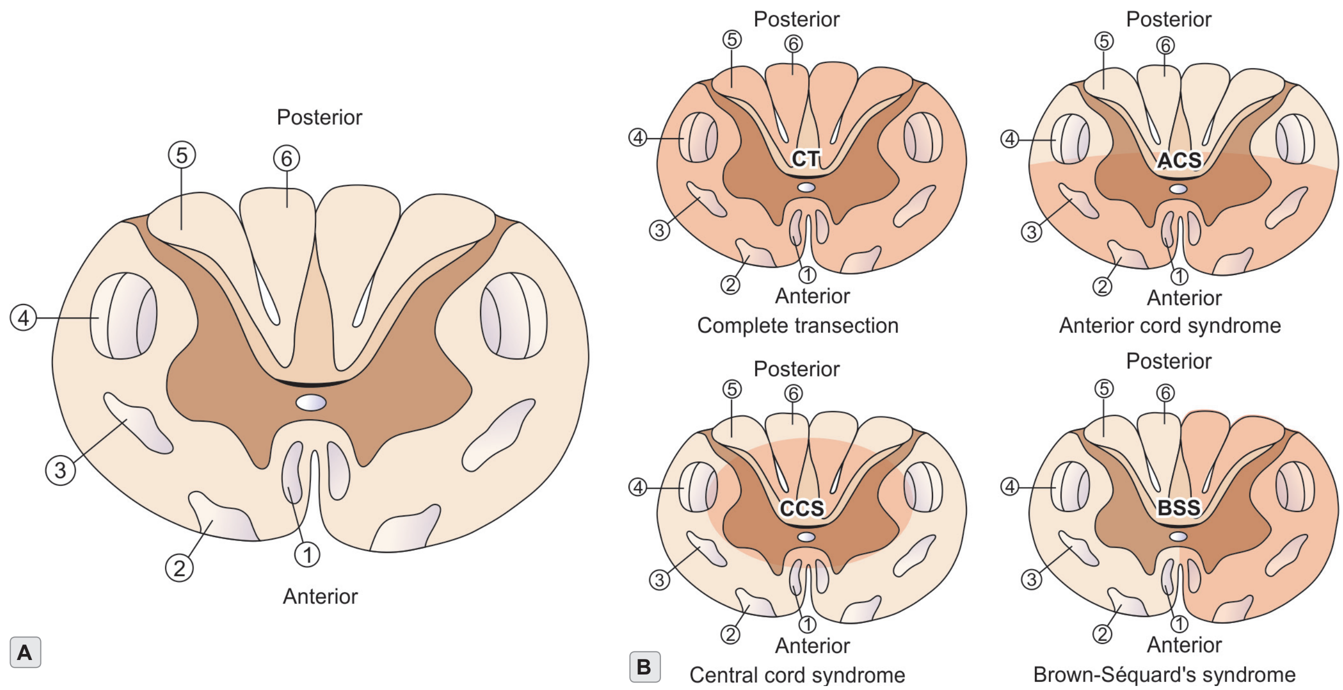
Incomplete transection of the cord results in various types of syndromes.

Anterior Cord Syndrome

In this, the *anterior portion* of the spinal cord is involved due to direct injury by a bony fragment of a fractured vertebra, prolapsed intervertebral disc or at times injury to the anterior spinal artery (loss of blood supply). Neurological involvement *manifests with complete motor loss, loss of pain and temperature discrimination below the level of injury, preserving (intact) deep touch, position and vibration sense (dorsal column sensations). Prognosis is poor.* Some sensory recovery can occur but motor paralysis is generally permanent.

Central Cord Syndrome

This was described by Schneider in the year 1954. It is commonly seen in cervical and upper thoracic spines. Here, the *central portion* of the spinal cord is involved due to compression of the cord between the vertebral body/osteophytes anteriorly and/or pinching by the buckled the ligamentum flavum posteriorly. *Hematomyelia* occurs followed by *destruction of the medial portion of spinothalamic and pyramidal tract (i.e the portion close to the center of the cord).* Upper extremities are more involved than the lower. Common in elderly with degenerative changes in the cervical spine (spondylotic spine). *Mechanism of injury is hyperextension.* Around 50% of the patients show functional recovery (Figs 6.12A and B).



Figures 6.12A and B

Cross-section of the spinal cord and the position of the different tracts in the spinal cord (A) and their involvement in complete and different types of incomplete cord syndromes (B) (refer text for details.); 1- Anterior corticospinal tract; 2- Anterior spinothalamic tract; 3- Lateral spinothalamic tract; 4- Lateral corticospinal tract; 5- Fasciculus cuneatus; 6- Fasciculus gracilis. (Upper limb portion is the most medial part nearer to the midline followed by the trunk and then the lower limbs). * Complete transection—CT; Anterior cord syndrome—ACS; Central cord syndrome—CCS; Brown-Séquard's syndrome—BSS.

Brown-Séquard's Syndrome

In this, *one-half of the cord is involved.* Manifests with *complete spastic paralysis and loss of dorsal column sensation on the ipsilateral side and loss of pain and temperature of the contralateral side, below the level of the lesion.* Functional recovery generally occurs and patients become ambulant.

Management of Spinal Injuries

Emergency Management

At the site of the incident: Attempt should immediately be made to immobilize the spinal column with the help of collars, braces and jackets if available. If not, the patient may be strapped to a wooden plank e.g. a detached door or a board and carried OR transported on a stretcher. Sand bags may be kept on either side of the neck during transportation if cervical spine injury is suspected, to prevent rotation of the neck.

When a spinal injury is suspected, the entire spinal column needs support. Hence, all movements should be prevented. The patient should be rolled and moved like a log of wood on to the stretcher. The method of shifting is known as log rolling method.

Medical Management

To be instituted as early as possible. Patient is kept nil orally.

Measures

i. Treatment of spinal shock with adequate intravenous fluids and vasopressors.
ii. Treatment of pain by giving analgesic injections.
iii. Catheterization of the bladder and recording of urine output.
iv. Prophylactic antibiotic therapy to prevent infection.
v. Methylprednisolone injection as a bolus dose of 1.5–2.0 gm is given at earliest if spinal cord injury is suspected to reduce the cord edema and further compromise.
vi. Assistance of breathing when required.
vii. Transportation to a center having facilities for the treatment of spinal injuries, at earliest.

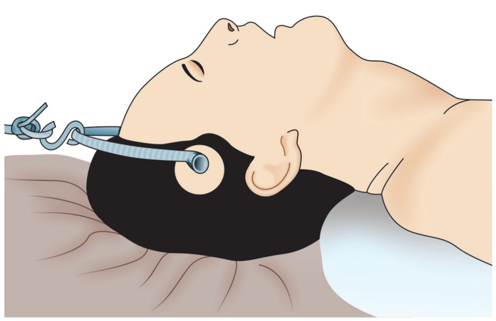
Definitive Management

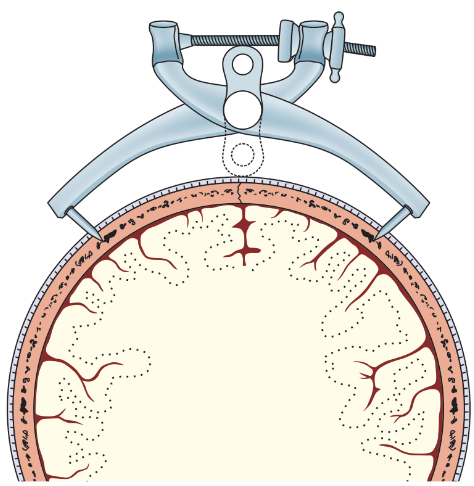
The definitive management starts only after initial stabilization of the general condition of the patient and after taking appropriate X-rays to assess the spinal column injury and MRI to assess in detail the nature of both column as well as the cord injury. Dynamic flexion/extension X-rays may be necessary when the static X-rays are normal but there is a high degree of suspicion of ligamentous disruption and presence of instability.

Definitive management depends on two factors:

a. Whether the injury is stable or unstable.
b. Whether there is cord injury or no.
   Loss of height of the vertebral body which is more than 50%, injury to posterior ligament complex and facet joints and presence of malalignment are the indicators of instability.

Nonoperative management: Indicated only in stable spinal column injury with irrecoverable cord injury or in those cases without cord injury. The treatment consists of immobilizing the spine (Figs 6.13A and B).

A


B


Figures 6.13A and B

Skull traction. Note that the sharp points of the tongs engage in the diploe and do not penetrate the inner table of the skull.

Types of collars and braces (Fig. 6.14)

a. For cervical spine
   Hard cervical collar, Extended cervical collar, Four post-cervical brace, Halo body orthosis/jacket, etc.
b. For thoracic and lumbar spine
   Thoracolumbar spinal brace, Halo pelvic traction, Bunion jacket, Thoracolumbar jacket, etc.

Operative management (Fig. 6.15): Indicated in all unstable spinal injuries with or without cord injury.

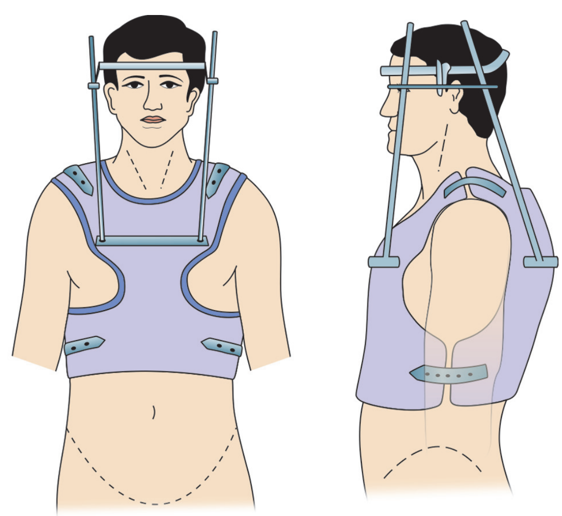


Figure 6.14

Halo body orthosis.

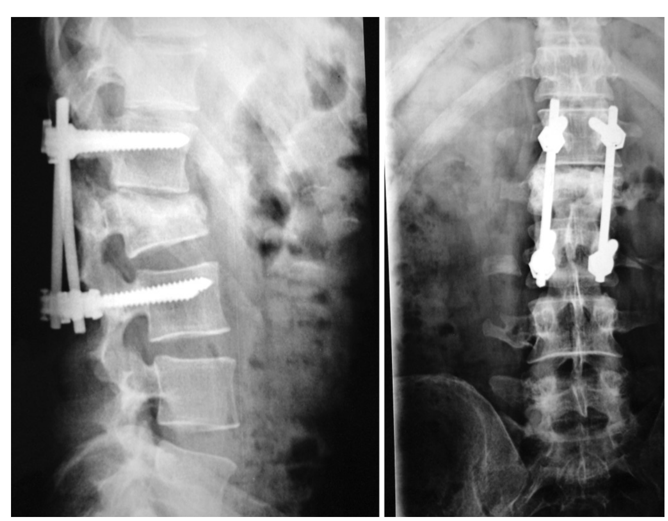


Figure 6.15

Fixation of a fracture dislocation at L1-L2 with pedicular screws and rods.

Aim: Is not only to stabilize the spine immediately but also to clear the compression on the cord (when present) and help in its recovery.

Early mobilization is possible after surgical stabilization which prevents all the complications of prolonged confinement to the bed, e.g. bed sores, hypostatic pneumonia, thromboembolism, etc.

Procedures

- Posterior decompression and instrumentation and fusion.
- Anterior decompression and fusion.
- Combined posterior and anterior instrumentation and fusion.

Management of Established Paralysis (Quadriplegia and Paraplegia)

Basic principles are to take care of:

a. The paralyzed bladder and the paralyzed bowel.
In the stage of spinal shock, there is flaccid paralysis of the bladder which needs catheterization for emptying of urine. Once, the spinal shock passes off a neurogenic bladder develops.
Types of neurogenic bladder (Fig. 6.16)
   i. *Automatic bladder:* This type of bladder is seen in complete transection of the spinal cord. In this, the bladder is devoid of inhibitory or facilitatory control from the higher centers in the brain. Hence, behaves independent of the higher centers. When the bladder is filled with urine it sends afferent sensory signals to lower centers of micturition situated in the S2, S3, S4 segments of the spinal cord which responds by sending efferent motor signals to the detrusor muscle in the bladder wall. The reflex arc becomes complete and the detrusor contracts powerfully resulting in complete emptying of the bladder. No residual urine is left in the bladder. Hence, there is less chance of developing complications of urinary tract infection and its sequel.
   ii. *Autonomous bladder:* This type of bladder is seen when the lesion affects the nerve roots after the spinal cord ends at the lower border of L1 e.g. conus medullaris and cauda equina lesion.
   The bladder is cut off from the lower centers of micturition situated in the S2, S3, S4 segments of spinal cord. No pathway is available for transmission of afferent sensory signals to the lower centers when the bladder is filled with urine. Hence, emptying has to occur only with the help of local myoneural stretch reflexes situated in the detrusor muscle. This emptying is less forceful and is incomplete. There is always residual urine in the bladder. Dribbling may occur. There is high chance of developing all the complications and sequel of urinary retention with recurrent urinary tract infection. Patient has to be taught to empty the bladder by "Crede's Maneuver", i.e. by applying gentle suprapubic pressure OR by intermittent self-catheterization (Fig. 6.17).

The bowel remains paralyzed in the stage of spinal shock and later starts functioning autonomously. Frequent spontaneous small quantity emptying is seen. Should be taken care of by good nursing.

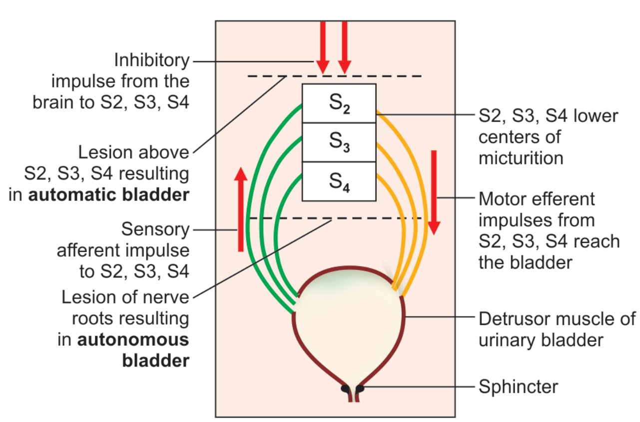


Figure 6.16

Normal mechanism of voiding of urine and the level of lesions responsible for the development of automatic and autonomous bladder.

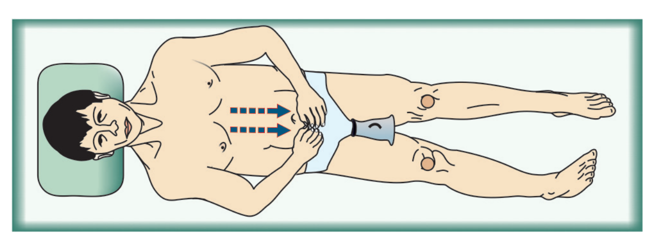


Figure 6.17

Crede's maneuver. Firm suprapubic pressure is applied to express the urine from the bladder.

b. To prevent other complications of prolonged confinement to bed e.g. pressure sore hypostatic pneumonia, thromboembolism, etc.
   Back care is a must to prevent pressure sore. Good nursing care, use of water bed or different types of pressure relieving mattresses will help in preventing pressure sores. These complications can also be prevented by early mobilization (Wheel chair) and exercise therapy by a well trained physiotherapist.

Signs of Grave Prognosis

If the cord transection is complete, the prognosis is poor. The person remains a quadriplegic or paraplegic through rest of his life. During the stage of spinal shock, there is flaccid paralysis with absence of perianal sensation and complete lumbar and sacral areflexia which may last for 24 hours to 7 days. Return of bulbocavernous reflex and anal wink which are normal cord mediated reflexes signifies the termination of the stage of spinal shock. Return of these reflexes alone in the presence of persisting paralysis, indicates grave prognosis.

Perianal Anesthesia (Fig. 6.18)

Persistence of anesthesia after recovery from spinal shock is an indicator of complete cord injury. Discrimination between sharpness and dullness when present, indicates incomplete injury.

Bulbocavernous Reflex (Fig. 6.19)

It is a polysynaptic reflex mediated through S1, S2, S3 segments of the spinal cord. The reflex is elicited by applying compression over the glans penis by squeezing or tugging on indwelling catheter at the same time monitoring reflex contraction of anal sphincter (Fig. 6.19).

Anal Wink

Otherwise known as anal reflex, perineal reflex, anocutaneous reflex is mediated through S1, S2, S3, segments of the spinal cord. The pudendal nerve carries these signals to the spinal centers.

The reflex is elicited by using a sharp pin and inducing a noxious tactile stimulus on the skin around the anal sphincter. This results in reflex contraction of the sphincter indicating the intactness of the reflex arc (Fig. 6.20).

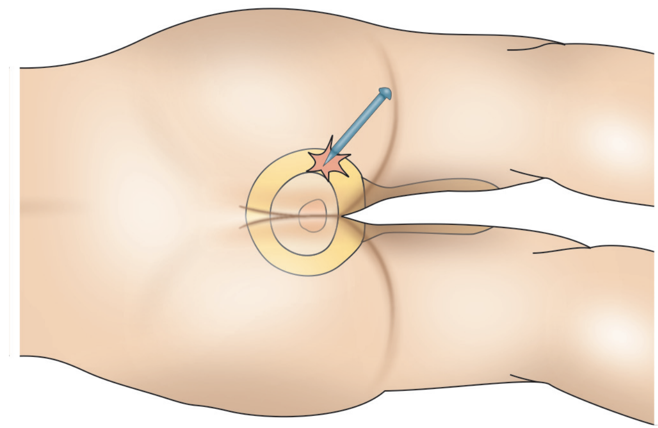


Figure 6.18

Diagrammatic representation of testing perianal anesthesia.

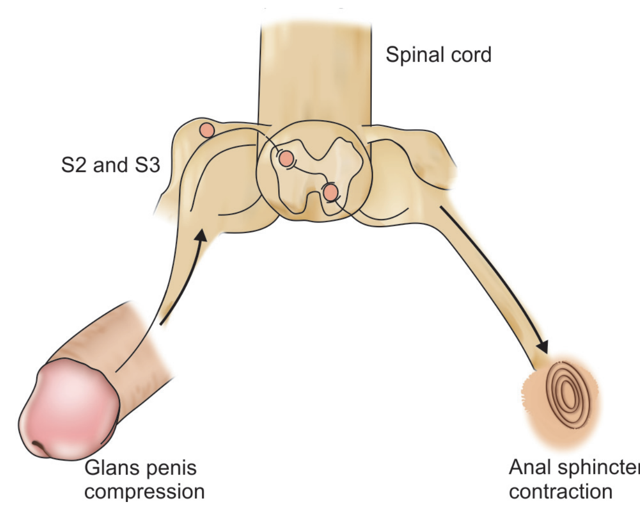


Figure 6.19

Diagrammatic representation of bulbocavernous reflex. Compression of glans penis will cause contraction of anal sphincter. Return of this cord mediated reflex indicates the termination of spinal shock and indicates grave prognosis.

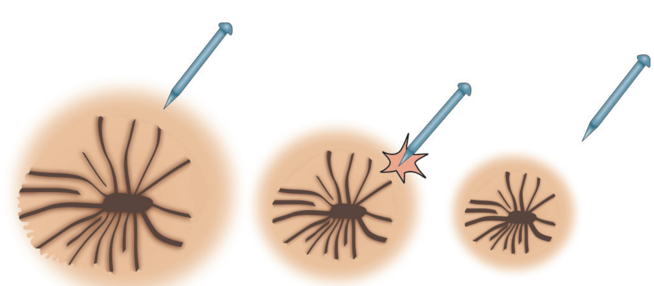


Figure 6.20

Anal reflex. Return of this reflex after termination of spinal shock in a person who remains as a quadriplegic indicates grave prognosis.

Interpretation

i. Absence of the reflex—stage of spinal shock.
ii. Return of the reflex—spinal shock has passed of and prognosis is grave.
iii. If spinal shock does not exist/not suspected but the reflexes are absent, these tests indicate that there can be conus medullaris or cauda equina lesion.

Rehabilitation

Centers for rehabilitation play a great role in helping paraplegics to lead an independent life. When necessary, assistance should be sought at earliest. The magnitude of injury is such that a bread earner for the family becomes a dependent for rest of his life. Psychological trauma is tremendous. Economic status is shattered. Hence, a paraplegic needs in addition to medical treatment, a good emotional support. Not only from family members but also from the medical team treating the patient. Rehabilitation of a quadriplegic is more challenging than a paraplegic. There is no greater satisfaction than to see these paralyzed people becoming vocationally rehabilitated, economically independent and emotionally stable.

A Ray of Hope for Paraplegics and Quadriplegics

On Friday the 23rd January 2009 FDA of United States of America, is said to have given permission for the first ever clinical trial on Embryonic Stem cell therapy for irrecoverable spinal cord injury.

Although animal experiments on rats had promising results as early as 2005, Stem cell therapy had acted as a double edged sword. On one hand, it had improved motor function below the level of the lesion. On the other hand, the rats developed greater pain sensitivity superior to it. Also, there existed a predictable possibility of stem cells growing into a tumor.

After much research, experimental study (multicentric) is currently being done on human beings by ***injecting Stem cells*** *14 days after the injury because of the possibility of stem cells getting destroyed in the process of inflammation occurring after injury if injected early. Too late an injection may not be effective because of degeneration occurring in the spinal cord and formation of scar tissue. The role of growth factors and other chemical mediators are also found to be crucial.*

Approximately 2 millions stem cells are to be injected in an attempt to generate oligodendrocyte progenitor cell that could ultimately form new myelin and develop capability to transmit signals from the brain down the spinal cord.

Experiments of transfusing human embryonic cord blood in spinal cord transected rats has also shown promising results.

However, concrete concepts are yet to be established and every one hopes that ***this dream turns out to be a reality in future.***

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7 Peripheral Nerve and Brachial Plexus Injuries

- *Introduction*
- *Structure of a peripheral nerve*
- *Response of a nerve to injury*
- *Classification of nerve injury*
- *Clinical symptoms, signs and tests useful in diagnosis*
- *Electrodiagnosis*
- *Management*
- *Pathomechanics of paralytic deformities and clinical signs*
- *Formation of brachial plexus*
- *Different injuries of brachial plexus*
- *Signs, symptoms, diagnosis and management*

PERIPHERAL NERVE INJURY

INTRODUCTION

Every peripheral nerve is a mixed nerve (unlike cranial nerves, which are either purely sensory, motor or mixed). Hence, injury to a peripheral nerve results in disturbance of sensory, motor and autonomic functions. As the spinal nerves exit through the spinal foramina they form plexus, i.e. the cervical, the brachial, the lumbar and the sacral plexus from which the peripheral nerves arise. Hence, peripheral nerves consist components from several nerve roots.

STRUCTURE OF A PERIPHERAL NERVE

The basic unit of a peripheral nerve is an axon. Axon is a direct extension of a nerve cell either from anterior horn cell, a dorsal root ganglion or a regional sympathetic ganglion. Each axon is covered by myelin and Schwann cell sheath. These axons with their sheaths are enclosed further in a delicate fibrous sheath known as endoneurium. A group or cluster of axons with their endoneurial tubes is known as a fasciculus or funiculus. This funiculus is enclosed in another sheath known as perineurium. Thus, a bundle is formed. These bundles of groups of axons are surrounded by a fairly thick fibrous sheath known as epineurium. Thus, each axon is covered and protected by three sheaths, namely, the endoneurium (the innermost), perineurium (the intermediate) and epineurium (the outermost) (Figs 7.1A and B).

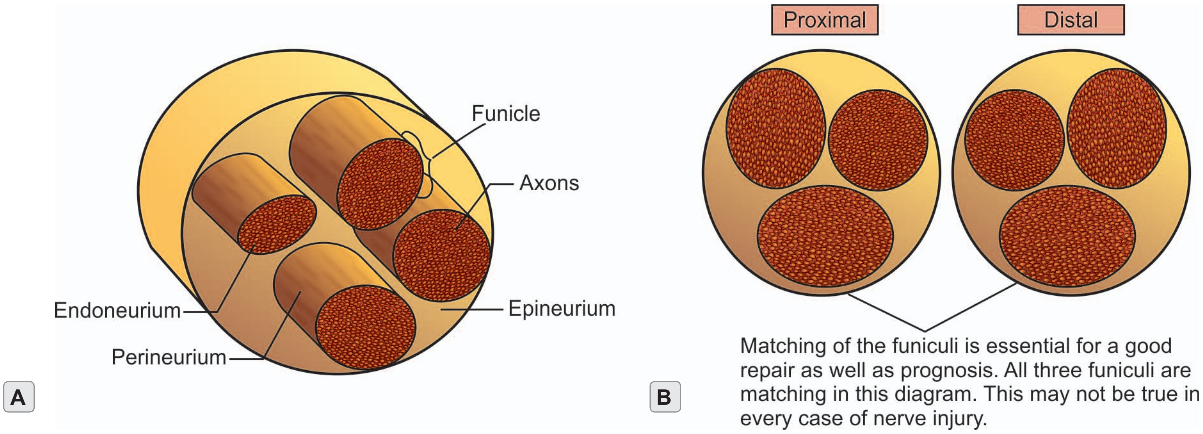
Note: *The number and disposition of the axons as well as the fascicles, vary from nerve to nerve. So is the thickness and the amount of neural sheath. Also, in the same nerve the structure varies at different levels. This is because the nerve branches out and the number of fibers become progressively less and less towards the terminal end of the nerve.*

RESPONSE OF A NEURON TO INJURY

The neuron disintegrates and degenerates following an injury. The time over which this process takes place, varies for sensory and motor fibers. It depends on the size and myelination of the nerve. The change that occurs *distal* to the injury is known as secondary

or *antegrade degeneration (Wallerian degeneration)* and the change that occurs *proximal* to the injury is known as *primary or retrograde degeneration.* Distal degeneration manifests in the form of clearing the dead tissue and emptying the tube and preparing it for receiving sprouting axonal buds, whereas the changes taking place proximally is to recoup and prepare the neuron to regenerate and grow. These changes take place over a period of 15 to 30 days (Fig. 7.2).

Lesser the disruption, greater is the recovery and return of function. Greater the disruption, lesser is the recovery and return of function.



Figures 7.1A and B

Structure of a peripheral nerve showing 4 Bundles (Funiculi enclosed in perineurium).

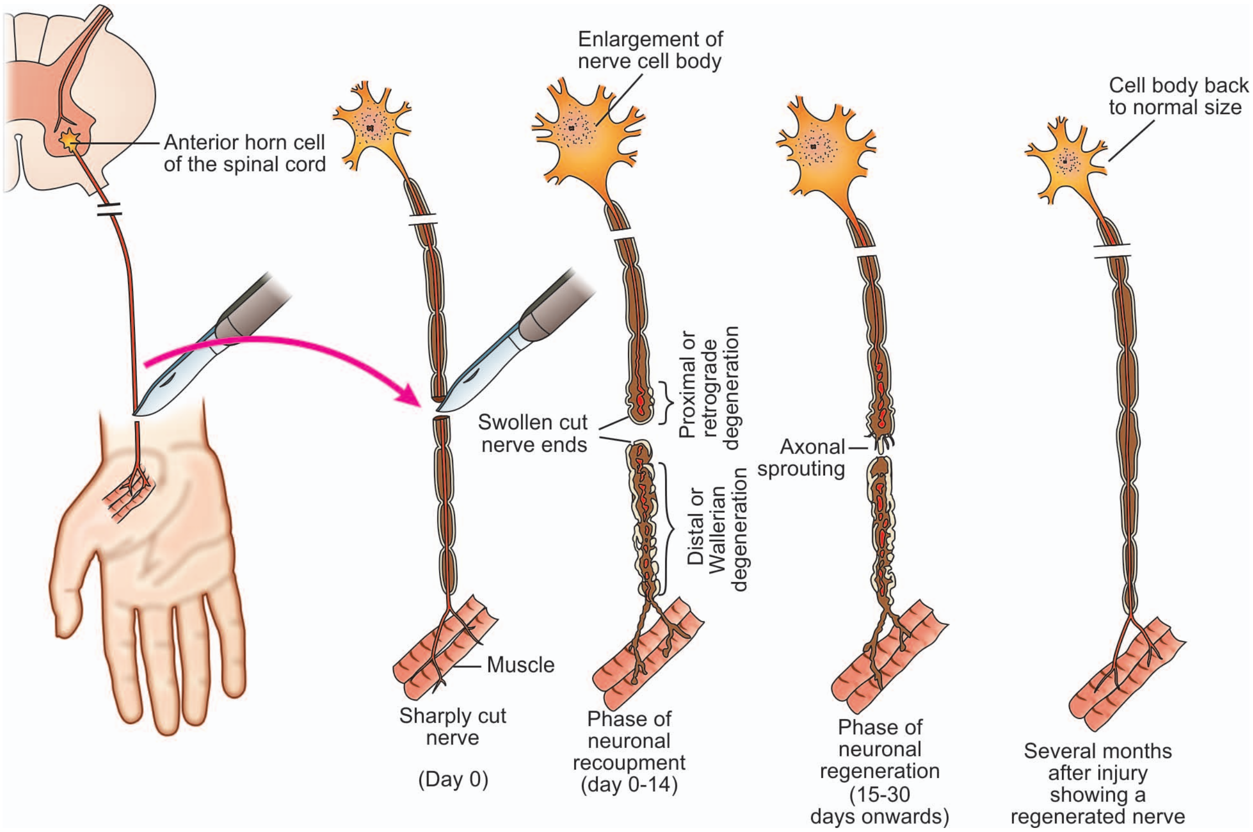


Figure 7.2

Response of a nerve to injury. Note the Wallerian degeneration occurring distal to the injury, proximal or retrograde degeneration occurring proximal to the injury. The process of regeneration begins only after a period of 14-21 days following injury.

CLASSIFICATION OF NERVE INJURY

Based on Seddon's Classification (1943)

Neuropraxia

In this, the injury is either contusion or compression of the nerve. There is a physiological block in conduction of the nerve impulse but the anatomical continuity is preserved. It is the result of transient ischemia. No Wallerian degeneration is observed. Recovery is complete, e.g. Saturday night palsy, crutch palsy.

Axonotmesis

In this the axons break but the sheath is preserved. Occurs as a result of traction and stretching of the nerve. Though wallerian degeneration occurs, good functional recovery is possible because of the intact nerve sheath, e.g. birth injury (except avulsion injury), tardy ulnar nerve palsy (Early).

Neurotmesis

It is the division of both, the nerve fibers as well as the nerve sheath. Seen when a sharp object cuts the nerve, which may be partial or complete. Hence, the terms partial and complete neurotmesis are used to describe the injury. Recovery is impossible without repair. Failure to recover and poor recovery are not uncommon. At times, needs secondary reconstructive procedures to gain useful function.

Based on Sunderland Classification (1951): (In brief)

I- Degree: Conduction of the nerve is interfered.	Neuropraxia of Seddon's
II- Degree: Continuity of the axon is interfered. III- Degree: Continuity of the endoneurial tube and its contents is interfered. IV- Degree: Continuity of the funiculus and its contents is interfered.	These represent varying grades of axonotmesis of Seddon's
V- *Degree:* Continuity of the entire nerve trunk is interfered (Disruption of the entire nerve).	Neurotmesis of Seddon's

VI- Degree of MacKinnon Dellon (1988): A combination of varying grades of Sunderland which co-exist in the same nerve. This means that a part of the nerve may have neuropraxia while the other part has axonotmesis.

Note: *Further,it should be remembered that a nerve may be partially cut by a sharp object resulting in partial neurotmesis. In such cases, the functional loss is proportionate to the degree of disruption. Some function may be preserved. Sensory and motor loss need not be complete. When such injuries are untreated they heal by forming a neuroma in continuity.*

DIAGNOSIS

Diagnosis is done on the basis of clinical evaluation of autonomic disturbance, sensory changes, motor changes and by performing relevant special tests. Sensory and motor loss develops immediately after injury. Autonomic disturbance though present after injury, takes some time to establish. Certain changes develop progressively over a period of time and are seen after a few weeks, e.g muscle wasting.

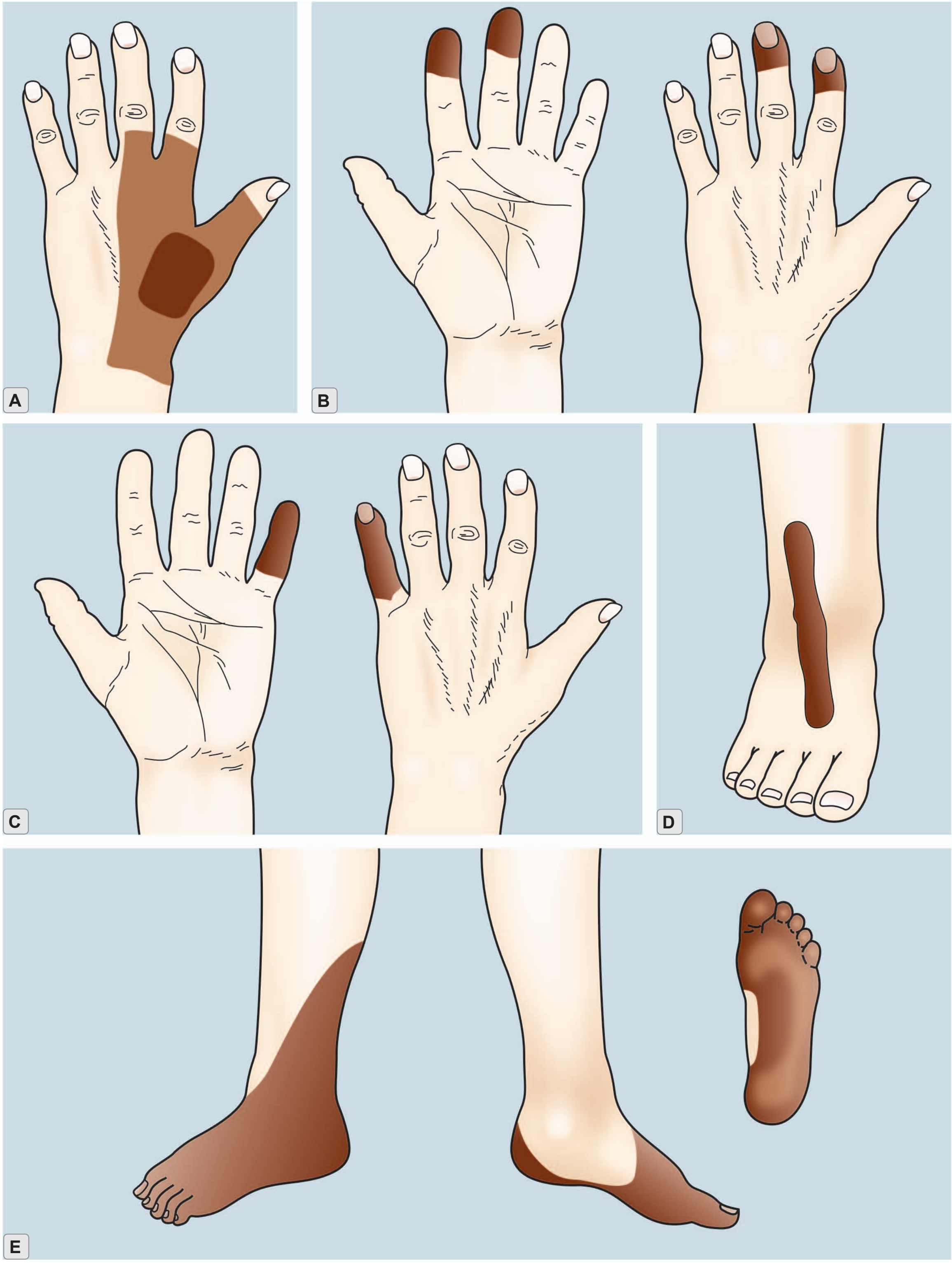
Note: *A scar/penetrating wound along the course of the nerve, especially at right angles, indicates the possibility of neurotmesis.*

Autonomic and Sensory Changes

a. Smoothness and dryness of the skin.
b. Atrophy of the pulp and conical tapering of the digits.
c. Brittle nails.
d. Loss of callosities and creases.
e. Absence of sweating.
f. Trophic ulcers.
g. Loss of sensation over the sensory area supplied by the peripheral nerve especially in the autonomous zone (Figs 7.3A to E).

Motor Changes

a. Paralysis of the muscles supplied by the peripheral nerve resulting in loss of function.
b. Pathognomonic deformities as a result of muscle imbalance.
   Median nerve: Injury at a higher level (at the elbow), results in both ape thumb and pointing index deformity. Injury at a lower level (at the wrist), results in only ape thumb deformity



Figures 7.3A to E

Autonomous zones of different peripheral nerves: (A) Radial nerve; (B) Median nerve; (C) Ulnar nerve; (D) Common peroneal nerve; (E) Sciatic nerve.

Ulnar nerve: Ulnar claw hand.
Combined ulnar and median nerve: Simian hand (hand of an ape)
Radial nerve: Wrist drop
Posterior interosseous nerve: Finger drop and thumb drop.
Sciatic nerve: Flail foot.
Posterior tibial nerve: Claw toes.
Lateral popliteal nerve: Foot drop.

These pathognomonic deformities when present are diagnostic of a peripheral nerve injury.

Special Tests (Discussed in Detail in Later Part of this Chapter)

a. Oschner's clasp test.
b. Pen test.
c. Card test.
d. Book test.
e. Igawa's sign.
f. Tinel's sign.

Sensory Zones of a Peripheral Nerve

Three zones are recognized. They are, the maximal zone, the intermediate zone, and the autonomous zone.

Maximal zone: It is the maximal area supplied by a peripheral nerve.

Intermediate zone: It is the area of overlap of the maximal zone of the different peripheral nerves.

Autonomous zone: It is the area exclusively supplied by a particular peripheral nerve (Figs 7.3A to E).

Note: *Sensory loss over the autonomous zone of a particular peripheral nerve, is the sure sign of that particular peripheral nerve injury. It is of immense help for a quick diagnosis. This is especially true in a case of polytrauma, where patient has fractures. In such a case, because of pain and loss of movement, it is difficult to examine in detail and elicit signs of a peripheral nerve injury.*

Diagnostic Tests

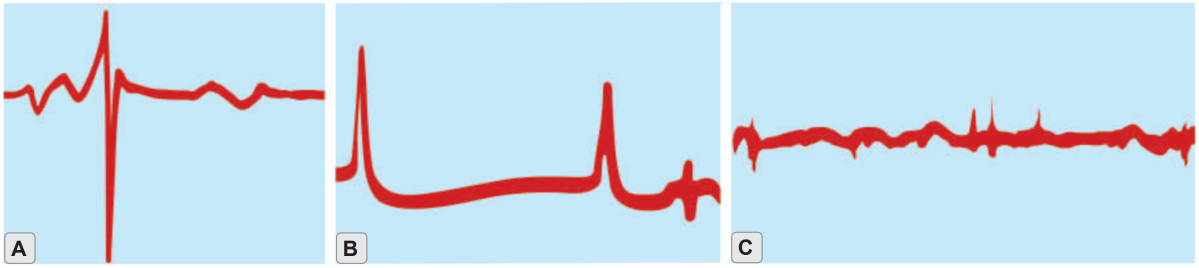
Electromyography (EMG)

The action potential generated in the muscle is recorded graphically both at rest and during activity (voluntary motor action potentials), by the insertion of a needle electrode. It is done at early and late intervals, as well as before and after nerve repair, compared and interpreted.

Denervation potentials: Every muscle is inherently vibrant with an electrical potential. This electrical potential remains masked as long as the muscle has an intact nerve supply. This is because, the impulse generated by a peripheral nerve is much greater than the resting inherent electrical potential of the muscle. When there is denervation, no impulse is transmitted through the peripheral nerve. Thus, it is possible to record this inherent electrical potential of the muscle. This is recorded initially, as sharp positive waves and later, as fibrillatory waves and are known as denervation potentials (Figs 7.4A to C).

Thus following conclusions are drawn:

a. Presence of denervation/fibrillation potentials indicate denervation.
b. Absence of denervation/fibrillation potentials as late as 3 weeks indicate intactness of nerve.
c. Absence of voluntary motor unit potentials indicate paralysis of the nerve.
d. Presence of voluntary motor unit potentials indicate intact functional nerve.
e. Polyphasic motor unit potential developing after denervation indicates reinnervation.



Figures 7.4A to C

Action potential during EMG study. (A) Electromyogram showing normal insertional activity; (B) Electromyogram showing positive sharp wave of denervation potential; (C) Electromyogram showing spontaneous fibrillation potentials of denervation.

Thus, on the basis of presence/absence of fibrillation potentials, voluntary motor unit potentials and polyphasic motor unit potentials, the status of the nerve after injury, can be assessed.

Strength Duration Curve

Uses the motor conduction property to assess the status of the injured nerve. As the name indicates, this is a curve plotted on a graph for different strengths and different durations of electrical stimulation within a physiological limit (Galvanic/Faradic current). The surface electrodes are used for stimulation.

The physiological limit of stimulation is expressed in terms of rheobase and chronaxie.

Rheobase

It is the minimal strength of the current, of infinite duration (e.g. 300 milliseconds/long duration), required to stimulate a muscle.

Chronaxie

It is the duration required to stimulate a muscle with the current strength of twice the rheobase. Chronaxie in a human skeletal muscle varies from 0.08 to 0.32 milliseconds.

A muscle with intact nerve, responds to any strength and any duration of current within this physiological limit of rheobase and chronaxie. The contraction is directly proportional to the strength and duration of the stimulus. A paralyzed muscle, does not respond to a stimulus of low strength of short duration (Faradic) but responds only to a stimulus of high strength and long duration (Galvanic). So, in a paralyzed muscle, the response is absent for a faradic stimulation but is present for a galvanic stimulation. Hence the curve plotted, smoothly ascends to the right of the normal curve. As regeneration takes place or in a partially injured nerve, the curve starts shifting to the left and a kink develops in the curve. And finally when the regeneration is complete the curve descends, becomes flatter and near normal (Fig. 7.5).

Starch Iodine Test (Tests Autonomic Function)

In starch iodine test, the area in question is first painted with iodine and kept dry. Then, starch powder is sprinkled and sweating is induced by covering the area with cloth OR making the patient drink a cup of hot coffee. If sweating is present, the color changes to purple indicating intactness of autonomous innervation. If absent, no color change is observed, indicating loss of autonomic function.

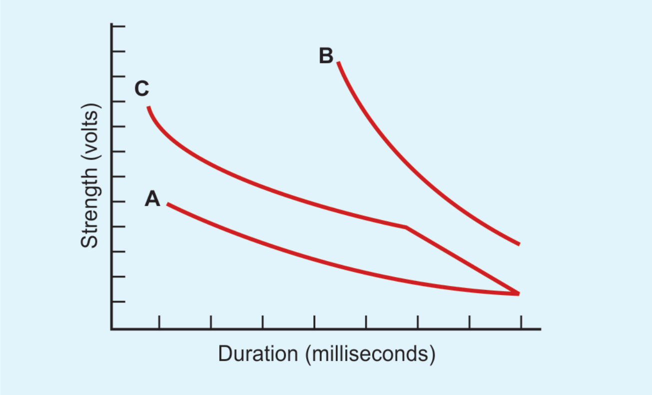


Figure 7.5
Showing changes in the SD curve during denervation as well as at regeneration. Strength duration curve (A) A normal nerve curve (or at complete reinnervation) (B) A curve seen in denervation (shift to the right) (C) Partial denervation/regeneration showing a kink in the curve.

MANAGEMENT

Neuropraxia and Axonotmesis

These injuries invariably recover and hence need only supportive measures. For example, Saturday night palsy, traction injury (not avulsion) of brachial plexus or any other peripheral nerve. The management is always nonoperative.

Measures undertaken are:

a. Drug therapy to relieve the edema and improve vascularity, e.g. anti-inflammatory drugs, steroids, etc.
b. Rest to the part by means of static splinting (prevents stretching) to facilitate healing.
c. Later, preventing contractures by means of dynamic splinting and exercise therapy.

Neurotmesis

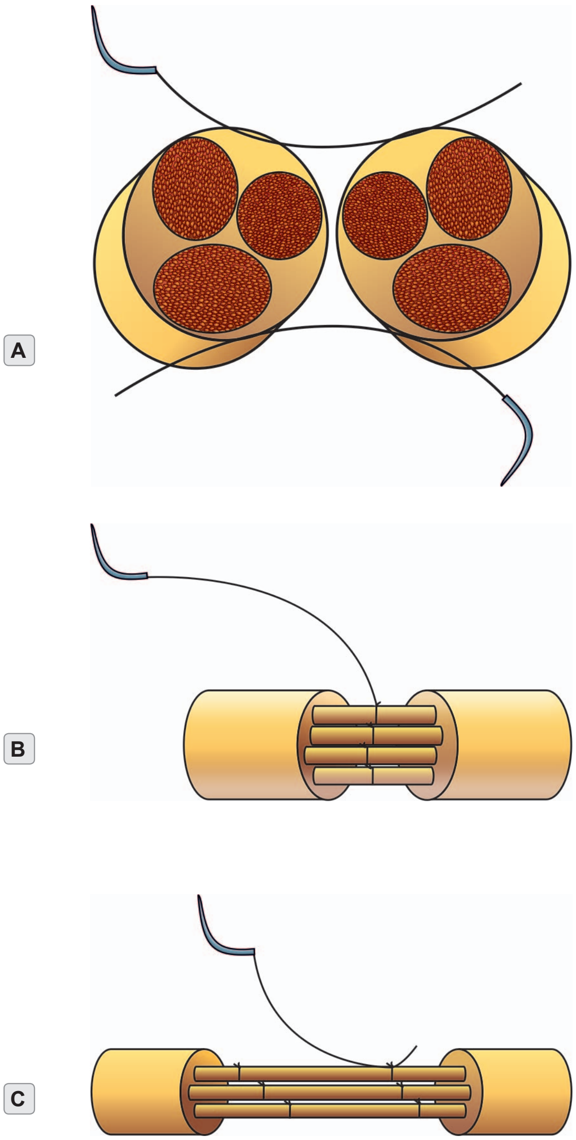
It is always treated by nerve repair, i.e. neurorrhaphy. Repair is done either immediately after the injury, which is known as *primary repair,* or at a later period after the initial wound heals, which is known as *secondary repair.* Repair is done by placing sutures on either side of cut epineurium or perineurium (nerve sheath) and approximating them. This joins the nerve.

During suturing, care should be taken to match and approximate the funiculus on either side of the cut ends of the nerve (Fig. 7.1B).

Nerve grafting: Nerve grafting is done only when there is loss of nerve tissue and when end to end repair by any means is not possible. Commonly, Sural nerve is used as the donor nerve (Fig. 7.7). Cadaveric nerve transplants are also in practice to bridge large defects.

Types of Nerve Repair (Neurorrhaphy)

a. *Epineurial repair:* Epineurium is used for placement of sutures (Figs 7.6A, 7.8 and 7.9).
b. *Perineurial repair:* Perineurium is used for placement of sutures (Figs 7.6B and C)
c. *Combined epiperineurial repair:* Both epiperineurium are used for placement of sutures.



Figures 7.6A to C

Different techniques of nerve repair shown diagrammatically. (A) Epineurial repair; (B) Perineurial repair; and (C) Nerve grafting.

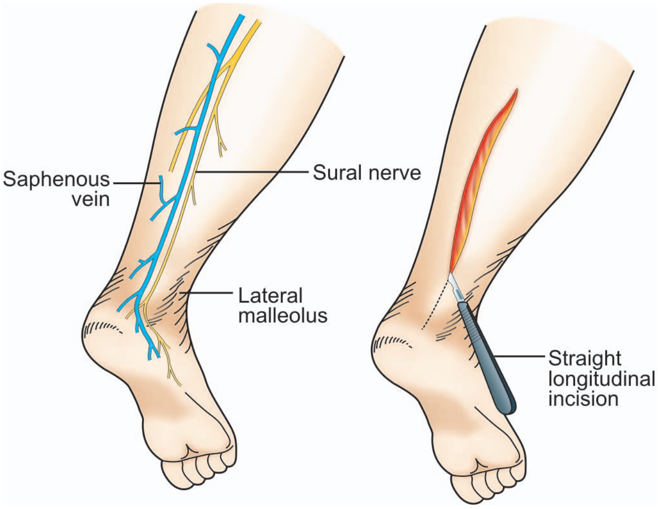


Figure 7.7

Method of obtaining sural nerve for nerve grafting procedure.

Figure 7.8

Primary epineurial repair of median nerve in the lower one-third forearm. Note the wound caused by sickle injury. It is in the midline along the course of the nerve.

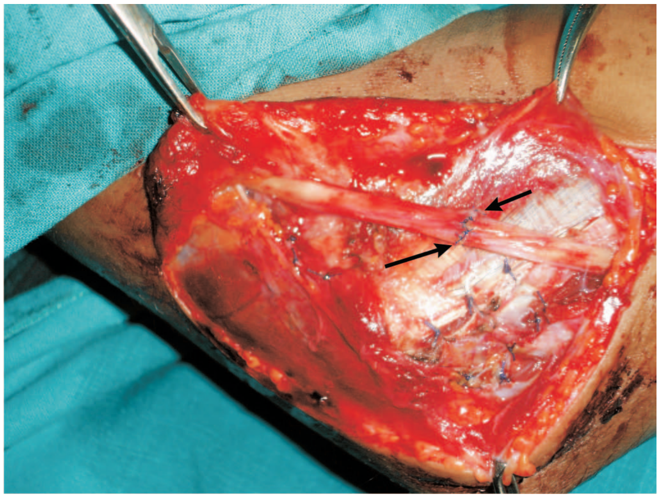


Figure 7.9

Primary repair of an ulnar nerve injury at the elbow. The nerve has been transposed anteriorly. Approximation of the fasciculi and epineurial nerve repair using 6-0 poly propylene sutures has been done. Good recovery was seen and patient regained useful grade of motor power with sensory recovery almost up to S3.

Factors Influencing Nerve Repair

a. *Age of the patient:* Younger the age, better is the result and vice versa.
b. *Duration of the injury:* Longer the duration, poorer is the result and vice versa.
c. *Level of the injury:* More proximal the injury, poorer is the chance of recovery and vice versa.
d. *Loss of the nerve tissue with defect:* Larger the defect, poorer is the result and vice versa.
e. *Nature of the cut ends:* Cleaner the cut ends better is the result when compared to ragged and lacerated cut ends.
f. Quality of repair.
g. *Technique employed:* Epineurial repair is always better than perineurial except when perineurial repair is better than epineurial. Direct end to end repair is better than nerve grafting except when nerve grafting is more desirable.

Methods of Closing the Gaps While Repair

a. Mobilization of the nerve.
b. Positioning of the joints.
c. Transposition or translocation.
d. Shortening of the bone (e.g. during reimplantation).
e. Sacrificing (dividing cutaneous branches selectively) not very important branches.

Critical Limit of Delay

It is that period within which if a nerve is repaired, some useful functional recovery can be expected. Beyond this period, no functional recovery is possible. This is because of irreversible fibrotic changes taking place both in the muscle and nerve tissue, distal to the injury and absence of functional tissue available for recovery and action.

The critical limit of delay varies from nerve to nerve and ranges from 6 to 15 months.

Motor and sensory grading in nerve injuries

Motor Grading:

Grade 0	No movement.
Grade 1	A flicker of movement.
Grade 2	Movement possible when gravity is eliminated.
Grade 3	Movement possible against gravity
Grade 4	Movement possible against gravity and resistance.
Grade 5	Normal power (Complete recovery).

Sensory Grading:

S 0	No Sensation.
S 1	Presence of pain sensation.
S 2	Presence of pain and some touch.
S 3	Presence of pain and touch with no overreaction.
S 3+	Presence of pain touch with two point discrimination.
S 4	Complete recovery (Normal).

Reconstructive Procedures in Cases of Absent Recovery

Tendon Transfers (Dynamic Procedure)

Tendon transfer is one among the reconstructive procedures. When origin of a tendon is left intact and the insertion is detached and shifted to a new location, the procedure is known as tendon transfer. Tendon transfers are done in peripheral nerve injuries in order to get some useful motor function, when there is no motor recovery. Transferred tendon substitutes the action of paralysed musculotendinous unit and provides some useful function.

Static Procedures

Basic principle in these procedures is to achieve a fixity at the site of deformity in a functional position (i.e. desirable position to facilitate maximum function). This fixity is achieved by shortening the soft tissues and causing contracture OR by fusing the joints. The procedures are as follows:

a. Dermodesis
b. Tenodesis
c. Capsulodesis
d. Arthrodesis

Only capsulodesis and arthrodesis are successful and are done, when indicated. Dermodesis and tenodesis stretch and fail over a period of time.

Correction of Claw Hand

Disability in a claw hand and the need for claw correction:

Among the three basic hand functions, namely, the grasp, pinch and the hook, the grasp is severely affected in claw hand. Pinch is affected to certain extent. The hook function, is preserved.

In a normal grasp, the hand first opens up fully thereby deepening the cup in order to accommodate an object which is to be grasped in the palm of the hand. Then, the fingers flex and close over the object. The flexion occurs first at the metacarpophalangeal joints and then at the interphalangeal joints. Thus, the fingers close over the object and the object is firmly grasped in the palm of the hand.

In claw hand, when a person opens up the hand for grasping an object, the metacarpophalangeal joints go into hyper-extension and the interphalangeal joints go into flexion. Thus, in the hand instead of a concavity developing at the MCP joints in order to deepen the cup, a convexity develops. Because of this, the object does not come into the palm of the hand. Further, when the person tries to close the hand over the object in order to grasp the object, the interphalangeal joints flex first followed by the metacarpophalangeal joints and the object slips out of an already closed hand (Figs 7.10A to E).

Principle of claw correction: The basic principle of claw correction is to prevent hyperextension at the metacarpophalangeal joints. This allows the long extensors mainly, extensor digitorum to act on the interphalangeal joints through their attachment to the extensor hood and extend them. Thus, clawing is prevented.

Tendon transfers for claw correction: Following tendons may be taken for claw correction.

- Extensor carpi radialis brevis/longus; **Paul Brand's procedure.**
- Extensor indicis and extensor digiti minimi; **Fowler's procedure.**
- Flexor digitorum superficialis of middle or ring finger; **Sterling Bunnel's procedure.**

Re-routing of the tendon (Fig. 7.11): Always re-routed volar to the transverse metacarpal ligament, through the lumbrical canal and attached to the extensor hood.

Static procedure for claw correction: Capsulodesis or capsuloplasty of Zancolli. In this, a flap of volar capsule of the metacarpophalangeal joints of the involved fingers is raised with a base distally. Then, with the finger in about 30° of flexion, the capsular flap is pulled and sutured proximally, onto itself. This produces a flexion contracture at the metacarpophalangeal joints of about 30° and prevents hyperextension. Alternatively, a small elliptical portion of the capsule is excised and the capsule is shortened and sutured to produce a 30° flexion contracture (Fig. 7.12). Thus, the extensor digitorum is now free to act on interphalangeal joints and bring about extension.

Opponens Plasty (Fig. 7.13)

This is a reconstructive procedure to restore opposition in irrecoverable median nerve injury. Following tendons may be taken.

- Flexor digitorum superficialis of middle or ring finger (Royle-Thompson's procedure and Sterling-Bunnel's procedure).
- Extensor indicis proprious.

Re-routing of the tendon: This is done by withdrawing the donor tendon proximally, to the level of the wrist crease and then making use of the distal part of palmar fascia or carpal ligament or flexor carpi ulnaris as a pulley, it is passed subcutaneously to the thumb. The attachment is to the neck of the metacarpal and the extensor pollicis longus or to the abductor pollicis brevis, hood of the thumb MCP joint and extensor pollicis longus.

Figures 7.10A to E

(A and B) Normal grasp. The hand opens up, the cup of the hand deepens to accommodate the object. Next, flexion at the metacarpophalangeal joints followed by flexion at the interphalangeal joints takes place and the object is firmly grasped. (C to E) Abnormal grasp. In a claw hand, the cup cannot form on opening up of the hand. Instead metacarpophalangeal joints hyperextend. Hence, the object is not accommodated in the hand. On closing, flexion at the interphalangeal joints occurs first, followed by at the metacarpophalangeal joints flexion. So, the object slips out of a closed hand and grasp function is thus affected.

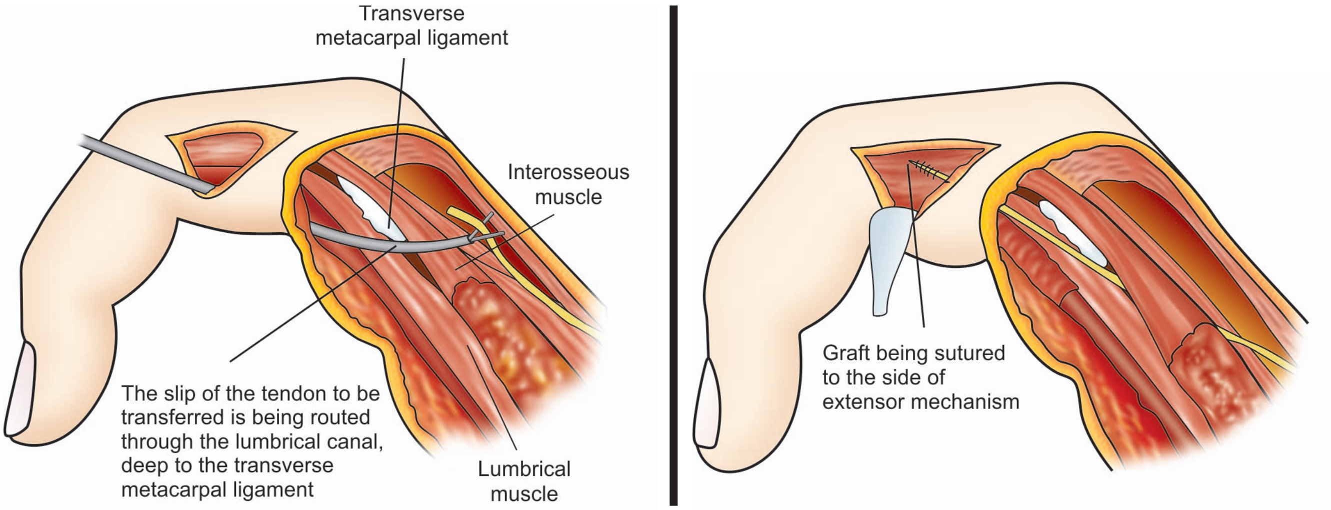


Figure 7.11

Re-routing of the tendon for correction of claw deformity.

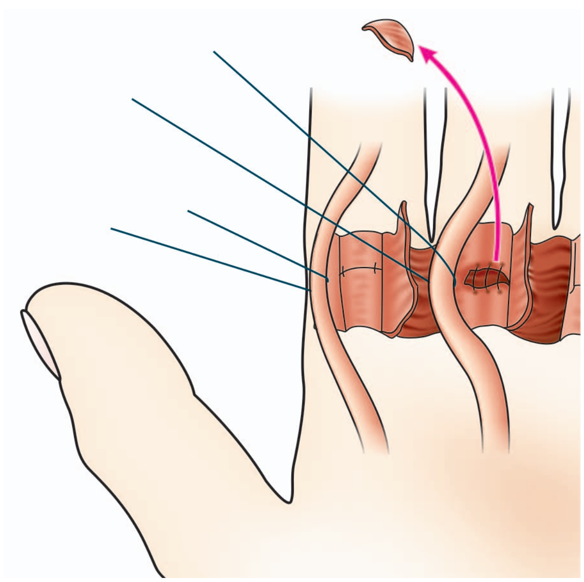


Figure 7.12

Diagrammatic representation of Zancolli's capsulodesis. A portion of the capsule of the MCP joint has been excised as shown in the diagram.When the remaining capsule is sutured, it produces a flexion contracture at the metacarpophalangeal joint and prevents extension.

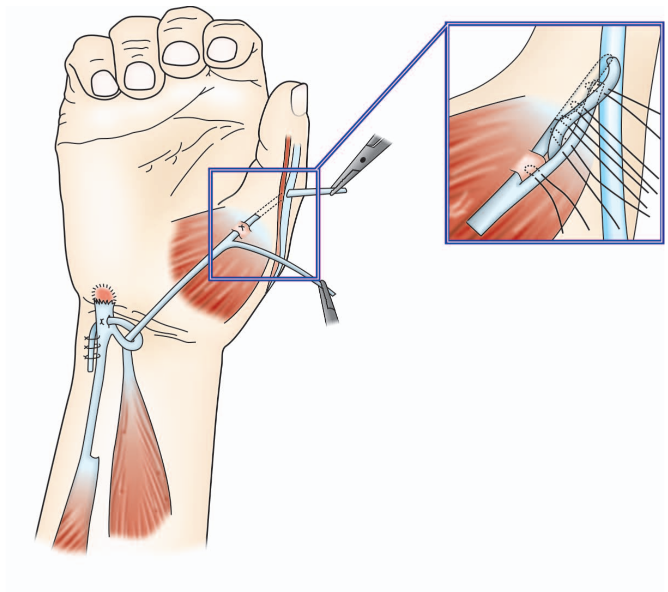


Figure 7.13

Opponens plasty based on Riordan's technique. Note a pulley which is made at the flexor carpi ulnaris and the method of attachment at the thumb. The transferred tendon is attached to APB, EPL and hood of the thumb MCP joint.

Tendon Transfer for Wrist Drop (High Radial Nerve Injury)

Functional deficit (Fig. 7.14) and substitution options:

a. *Wrist extension:* Pronator teres to extensor carpi radialis longus and brevis.

b. *Finger extension:* Flexor digitorum superficialis (iii)/ flexor carpi ulnaris/flexor carpi radialis (any one of the three) to Extensor digitorum.

c. *Thumb extension:* Flexor digitorum superficialis (iv)/ palmaris longus (any one of the two) to Extensor pollicis longus.

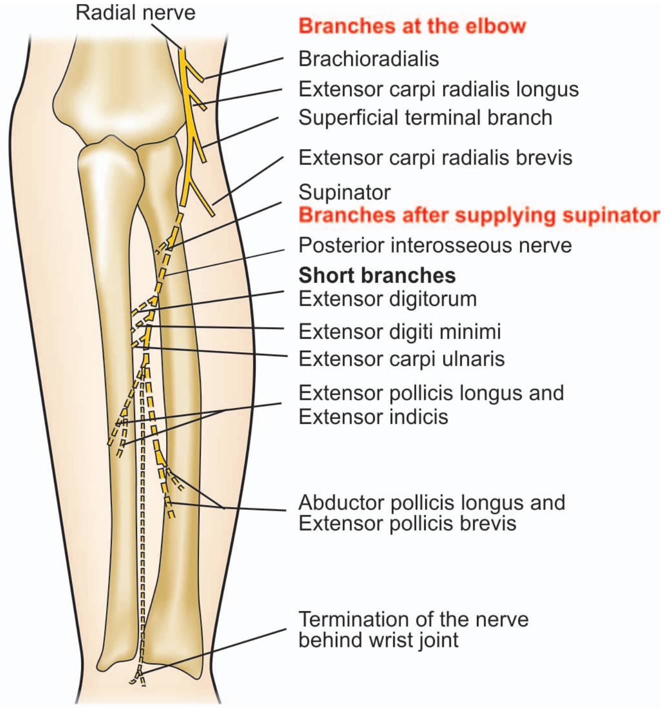


Figure 7.14

Muscles supplied by the radial nerve and the posterior interosseous nerve.
Note that the brachioradialis, extensor carpi radialis longus and brevis receive their nerve supply from radial nerve and so are spared from being paralyzed when posterior interosseous nerve is injured.

Tendon Transfer for Finger and Thumb Drop (Low Radial Nerve Injury)

Functional deficit and substitution options

a. Finger extension: Brachioradialis/flexor digitorum superficialis (iii) to Extensor digitorum.

b. Thumb extension: Palmaris longus/flexor digitorum superficialis (iv) to Extensor pollicis longus.

Tendon Transfer for Foot Drop

a. *Ober's procedure:* Tibialis posterior is transferred and inserted to base of the third metatarsal/ III cuneiform (subcutaneous tunnel—circumtibial route)

b. *Barr's procedure:* Tibialis posterior is transferred and inserted to III cuneiform (through the interosseous membrane—interosseous route).

Tendon transfers for foot drop are non phasic transfers and need accurate postoperative management for phasic conversion.

Figure 7.15

Method of assessing the power of tibialis posterior tendon both against gravity and resistance. Note that the subject is made sit on an examination table and cross the limb over the other and then asked to invert the foot against resistance applied to the forefoot, by the examiner. The tibialis posterior stands out prominently, just behind and little above the medial malleolus (see red arrow).

Prerequisites for a Tendon Transfer

a. The tendon chosen for transfer should have power of grade IV or more (Fig. 7.15).
b. Agonists are preferred to antagonists.
c. Joints on which the tendon acts should be supple.
d. Range of excursion of the transferred tendon should be almost similar to that of paralyzed tendon.
e. Line of action should be as straight as possible.

Tendon transfers recommended for different nerve injuries is summarized in Table 7.1 at the end of the chapter.

Injuries of the Other Peripheral Nerves

Spinal accessory nerve: Gets injured as a result of penetrating injury and accidental injury during surgical procedures (e.g. cervical lymph node biopsy).

It is a motor nerve supplying trapezius muscle. Hence, paralysis results in rotation of the scapula in a distal and lateral direction drawing the inferior angle closer to the midline. This results in not only in the weakness of the shoulder girdle but also in the inability to lift the shoulder beyond 90°. The winging of the inferior angle seen in this, disappears on raising the arm anteriorly unlike the winging seen in serratus anterior paralysis.

Suprascapular nerve: Commonly involved in penetrating injuries.

It supplies the supraspinatus, infraspinatus and the teres minor and gives an articular branch to the shoulder joint. Injury, results in paralysis of these muscles manifested by wasting and limitation of the movement of the shoulder.

Long thoracic nerve C5 C6 C7: Commonly injured in a penetrating injury or by means of a traction force in the angle of the shoulder and the neck.

As it supplies the serratus anterior muscle the classical winging of the scapula is seen. This is demonstrated by asking the patient to push the wall with both upper limbs raised forwards.

Axillary nerve C5 C6: It is injured by a direct penetrating injury or as a result of fractures, dislocations and fracture dislocation around the shoulder.

Results in weakness of abduction because of the paralysis of the deltoid muscle. Sensory loss when present, is over the deltoid region.

Musculocutaneous nerve C5 C6: It is injured by direct penetrating injuries and in anterior dislocation shoulder. Though it supplies the biceps brachii, coracobrachialis and brachialis, the injury is often missed because

i. Only biceps is amenable for testing.
ii. Pain of injury prevents testing.

Femoral nerve L2 L3 L4: It is commonly injured by means of a penetrating injury.

Often missed early and diagnosed late because of:

a. Alarming hemorrhage due to associated vascular injury.
b. Presence of extension because of active functioning tensor fascia lata, gracilis, gluteus maximus and gastrocnemius (This is misleading).
c. Small autonomous zone which is situated superior and medial to patella.

Symptoms and signs: Become evident after some time. They are:

a. Decreased sensation in the thigh, knee, or leg.
b. Numbness, tingling, burning over the thigh.
c. A feeling of the knee 'giving way' or buckling.
d. Weakness of the hip flexion knee/extension including difficulty going up and down stairs—especially down.
e. Atrophy of the thigh.
f. Loss of knee jerk.

Investigations like EMG and NCV, help in the diagnosis.

Sciatic nerve: Injured by penetrating injury, fracture dislocations around the hip and fracture shaft of the femur. Autonomous zone includes, almost the entire foot except a small area on the medial aspect in the mid foot region (Refer Fig. 7.3E). Functional loss is severe as there is paralysis of all the muscles of the posterior compartment of the thigh and muscles of the leg and the foot. The results of repair are poor because the regenerating axon has to travel a long distance to reach the motor end plate. Hence, takes a long time. By then, the motor end plates as well as the muscles would have atrophied, even if there had been an attempt to keep their activity by regular electrical stimulation and other physiotherapeutic measures. This is especially true when repaired late. Beyond 12 to 15 months, i.e. beyond critical limit of delay, repair should never be attempted.

Management

Principle of management of these nerve injuries remain the same (except sciatic nerve).

As the injury involving the above nerves is not very common, the functional loss not very severe and patients generally accept the resulting deficit easily, not much information is available regarding the repair of these nerves. However, reconstructive procedures are described when deficits warrant intervention.

A general layout for handling cases of nerve injuries is shown in Flow chart 7.1.

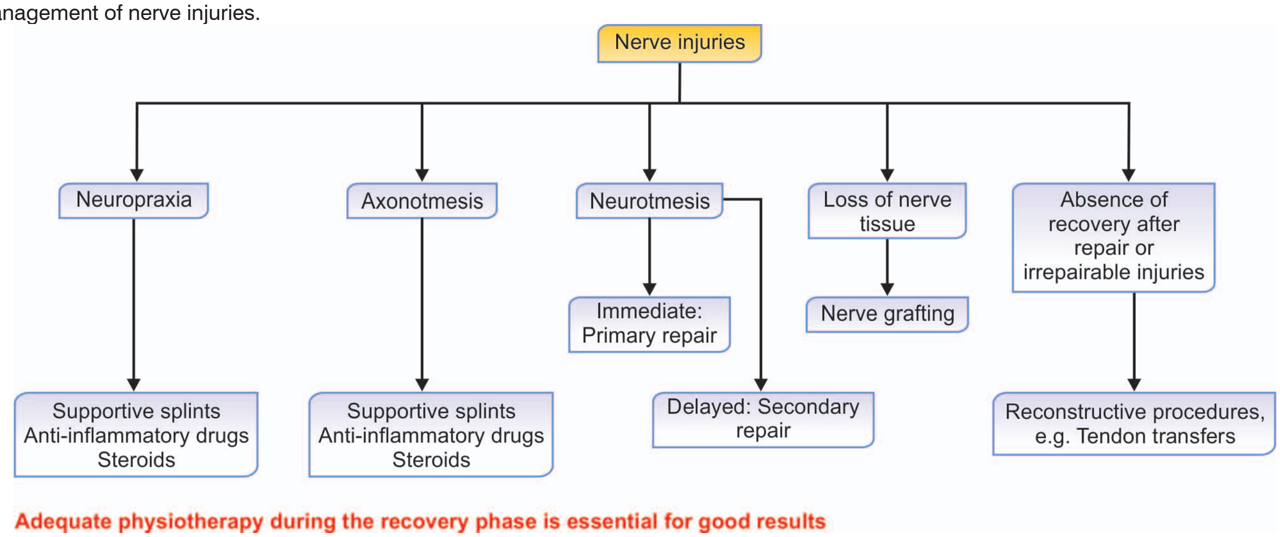
Pathomechanics of Various Deformities and Signs

Claw Hand (Intrinsic Minus Deformity) (Fig. 7.16)

Claw hand is defined as a dynamic deformity characterized by *hyperextension at the metacarpophalangeal joints and flexion at the interphalangeal joints*. If only ulnar nerve is involved, the deformity of *ulnar claw hand* or partial claw hand develops. If both ulnar and median nerves are involved (low ulnar and low median; high ulnar and low median), the deformity of *total or combined claw hand*, otherwise called the 'Simian hand' develops.

At every joint in the hand, there is a balance, maintained by the action of flexors on one side and the extensors on the other. If one is paralyzed, the joint moves in the other direction. *At the metacarpophalangeal joints*, the flexor is the lumbrical and extensor is the extensor digitorum. *At the proximal interphalangeal joints*, the flexor is the flexor digitorum superficialis and

Flow chart 7.1:
Management of nerve injuries.



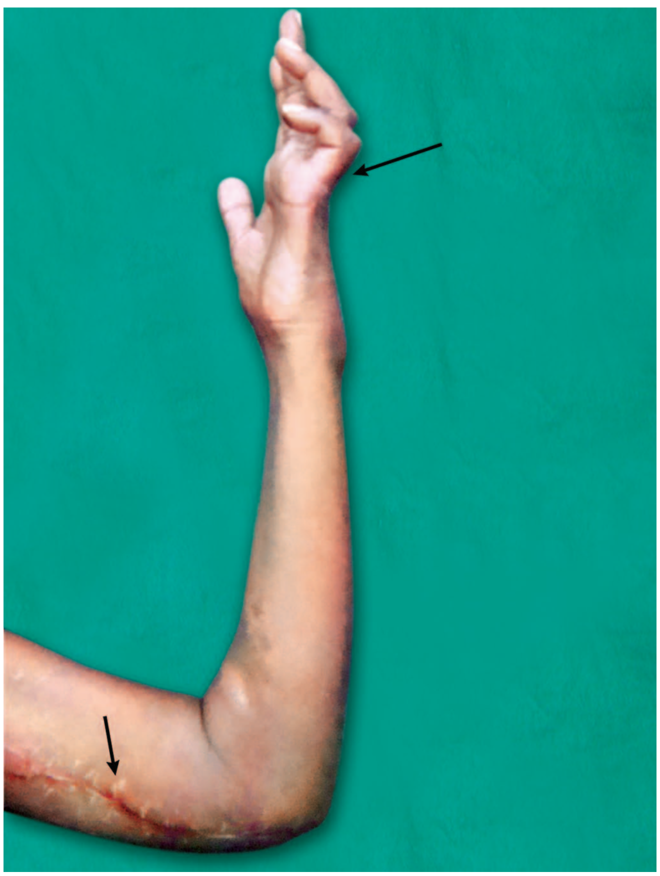


Figure 7.16
A classical 'Ulnar claw' hand with hyperextension at the metacarpophalangeal joints and flexion at the interphalangeal joints of ring and little fingers only. Hypothenar muscle atrophy is pronounced. Note the scar at the elbow along the course of the nerve (marked by an arrow) which indicates possibility of neurotmesis of the nerve.

extensor is the extensor expansion (also known as dorsal digital expansion, extensor hood, extensor apparatus, extensor aponeurosis). *At the distal interphalangeal joints*, the flexor is the flexor digitorum proundus and the extensor is the extensor hood (Figs 7.17 and 7.18).

In ulnar nerve injury, there is paralysis of all the interossei and the medial two lumbricals. Therefore, at the interphalangeal joints, extension is lost and the joints are subjected to unopposed action of flexor digitorum superficialis and or profundus. This causes flexion at these joints. Whereas, at the metacarpophalangeal joints, flexion is lost and the metacarpophalangeal joints are subjected to unopposed action of extensor digitorum resulting in hyperextension of the joints. Hence, deformity of ulnar claw develops (Fig. 7.16).

In combined low ulnar and low median nerve injury, there is paralysis of lateral two lumbricals in addition. Hence, the deformity of combined claw hand involves the lateral two fingers as well. The pathomechanics is the same as that of ulnar claw. The thumb remains by the side of other fingers because of paralysis of adductor pollicis and thenar muscles thereby subjecting it to unopposed action of thumb extensors and the long abductor. Hence, the hand resembles the hand of an ape and is known as 'simian hand'.

In combined high ulnar and median nerve injury, all the flexors are paralyzed. Hence, classical claw deformity is not seen except for hyperextension at the metacarpophalangeal joints. The flexion deformity at the interphalangeal joints is minimum.

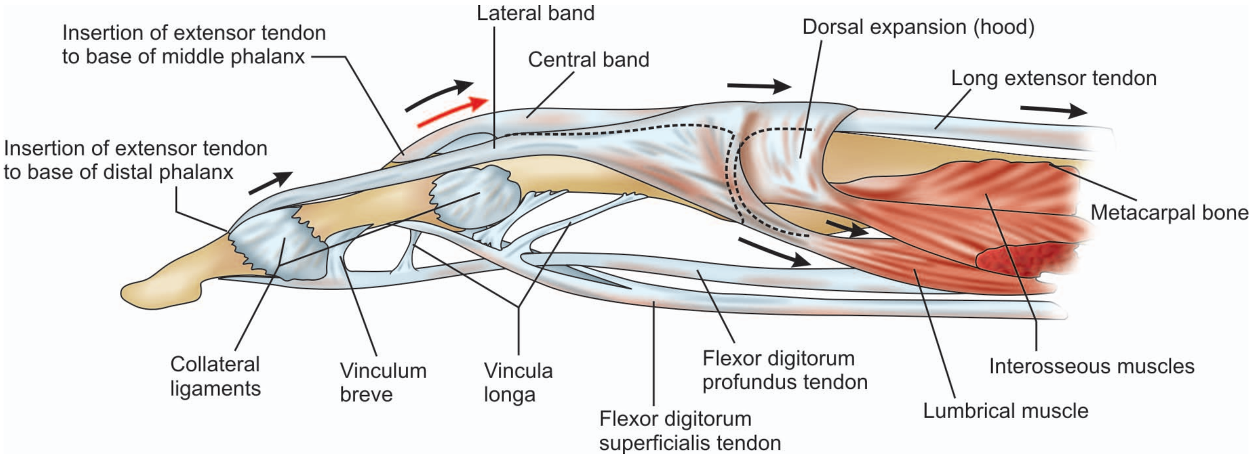
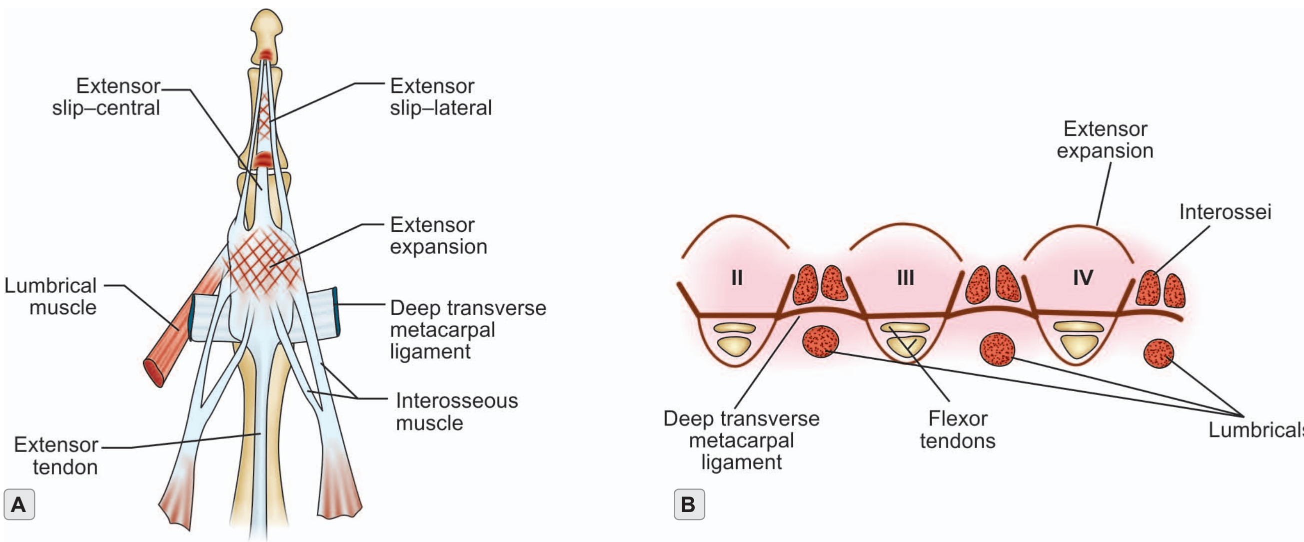


Figure 7.17

Balance at the joints of the hand is maintained by the flexor muscles on one side and the extensor muscle on the other.



Figures 7.18A and B

(A) Extensor expansion and (B) the relation of interossei, lumbricals, extensor expansion and flexor tendons at the level of the MCP joint.

Note: *This classical pathomechanics applies to low ulnar as well as combined low ulnar with low median nerve injury. Partly applies to combined low ulnar with high median nerve injury. It does not apply to combined high ulnar with high median nerve injury.*

Ulnar Paradox

Higher the lesion, lesser is the deformity. Lower the lesion, greater is the deformity. This is because, in high ulnar nerve injury, of the two finger flexors—only flexor digitorum superficialis is intact and acting (because it is supplied by the median nerve) and flexor digitorum profundus is paralyzed. Hence, deformity of finger flexion is less pronounced. Whereas, in low ulnar nerve lesion, the flexor digirum superficialis and the flexor digitorum profundus both are intact and acting. Hence the deformity is of finger flexion more pronounced.

Ape Thumb Deformity

Normally, the thumb is placed at right angles to the other fingers. When there is median nerve injury, the thumb remains in line with the other fingers, resembling the thumb of an ape. This is because of the unopposed action of adductor pollicis and extensor pollicis brevis which are supplied by ulnar and radial nerves respectively (Fig. 7.19).

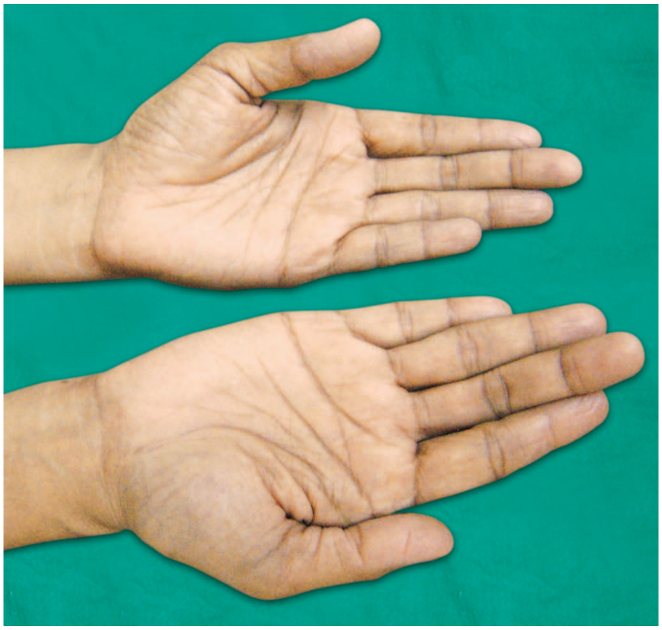


Figure 7.19

Normal left hand where the thumb is placed at right angles to the other fingers and the right hand with 'Ape thumb' deformity where the thumb is remaining parallel by the side of the other fingers.

Wrist Drop (Fig. 7.20A)

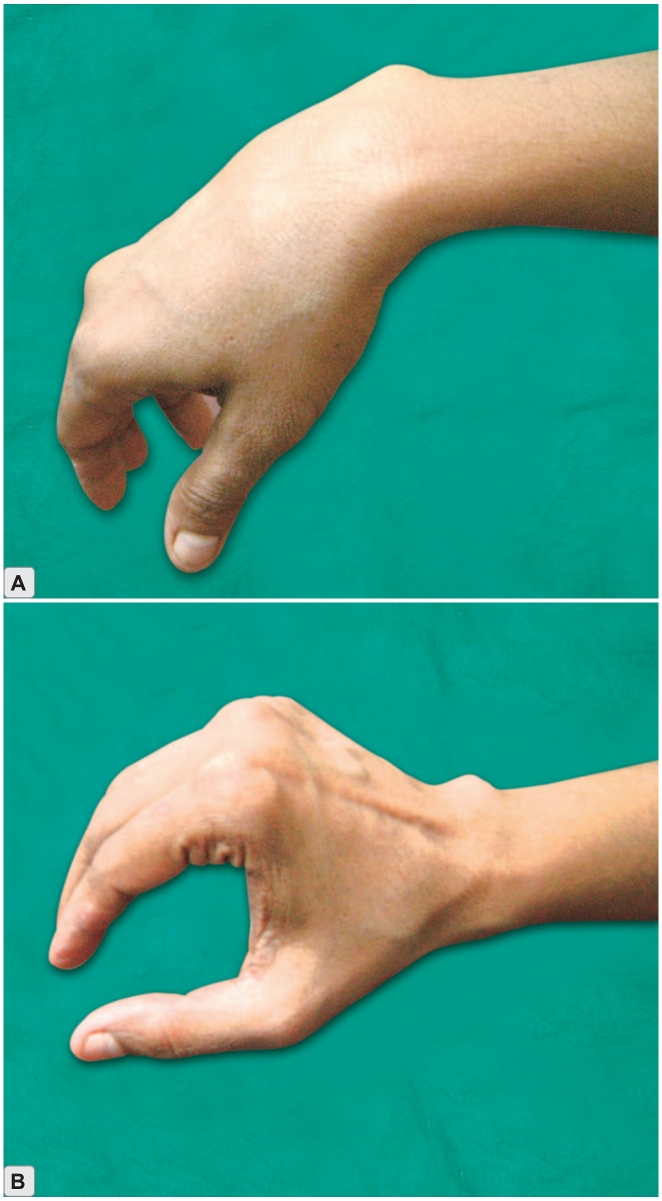
Is the inability to dorsiflex the wrist, fingers and the thumb, actively.

It results because of the paralysis of all the extensor muscles of the forearm as a result of radial nerve injury. The muscles paralyzed are:

a. Abductor pollicis longus and extensor pollicis brevis
b. Extensor carpi radialis longus and brevis
c. Extensor pollicis longus
d. Extensor digitorum and extensor indicis
e. Extensor digiti minimi
f. Extensor carpi ulnaris
g. Brachioradialis
h. Supinator.

Finger Drop and Thumb Drop

Seen in posterior interosseous nerve injury. Except brachioradialis, supinator, extensor carpi radialis longus and brevis, all the other muscles of the extensor compartment of the forearm are paralyzed. Hence, wrist extension is possible but not the finger and the thumb extension (Fig. 7.20B).



Figures 7.20A and B

Classical attitude of (A) Wrist drop and (B) Finger drop.

Flail Foot

Seen in sciatic nerve injury because of paralysis of both dorsiflexors and plantar flexors of the foot (All the muscles in the leg and in the foot are paralyzed).

Claw Toes

It is seen in posterior tibial nerve injury in the region of the ankle. Deformity occurs because of paralysis of the intrinsic muscles of the foot.

Foot Drop

Seen in lateral popliteal nerve injury because of paralysis of dorsiflexors of the foot. The muscles paralyzed are:

a. Tibialis anterior
b. Extensor digitorum longus
c. Extensor hallucis longus
d. Peroneus longus and brevis
e. Peroneus tertius
f. Extensor digitorum brevis.

The classical gait seen in foot drop is the 'High stepping gait'. Normal gait is a 'heel to toe' gait whereas, the high stepping gait, is a 'toe to heel' gait. This is because of the paralysis of the dorsiflexors. In this gait, the heel strike of stance phase of gait cycle is lacking. Hence to avoid injury, patient lifts the leg up and brings it down to the ground. Thus, toes touch the ground before the heel resulting in a 'High stepping' gait (Refer chapter on Gait for details).

Oschner's Clasp Test

This is a test employed to diagnose high median nerve injury. It is performed by asking the patient to clasp the hands. When there is a high median nerve injury, the index finger remains straight and points forwards (pointing index), indicating paralysis of flexor digitorum superficialis and flexor digitorum profundus. The test is positive when there is a pointing index.

Pathomechanics: Though the median nerve supplies lateral two profundi, the middle finger does not point even though it is paralyzed. This is because the middle finger profundus takes origin from the common aponeurosis of the medial two profoundi which are supplied by the ulnar nerve. Hence, when the medial two profundi (which are supplied by the ulnar nerve) contract, to some extent, the middle finger profundus also contracts (Fig. 7.21) and hence does not point.

Pen Test

This is a test employed to test the abductor pollicis brevis muscle. The test is done by asking the patient to place the hand flat on the table with the palm facing upwards and keeping a pen at some distance over the thumb. Next, he is asked to touch the pen with his thumb. If the patient is able to touch the pen with his thumb abducted, the test is negative. If the patient is not able to touch the pen, the test is positive.

Figure 7.21

Positive Oschner's clasp test and the pointing index *sign.* Seen only in high median nerve injury.

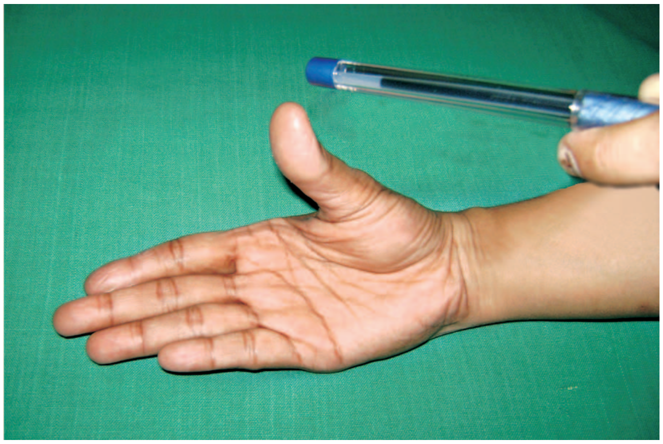


Figure 7.22

A negative pen test. The person is able to touch the pen using his abductor pollicis brevis. Positive sign (not able to touch the pen) is seen in both high and low median nerve injury.

The test is positive both in high and low median nerve injuries (Fig. 7.22).

Card Test

This is a test employed to test the interossei muscle. The function of the palmar interossei is adduction and the dorsal interossei is abduction. The test is performed by asking the patient to hold a card which is inserted between the two fingers. During the process, first the finger abducts and then, while gripping the card it adducts. The test is said to be positive when the patient is not able to grip the card between the fingers (i.e. the fingers neither abduct nor adduct) and the card is easily

pulled out by the examiner, who uses a similar grip to pull out the card (Fig. 7.23). If the patient is able to hold the card, the test is negative.

Book Test and Froment's Sign

It is a test employed to test the adductor pollicis muscle. The patient is asked to hold a book in the 1st web space. The examiner holds the same book in a similar manner and tries to pull out the book. When adductor pollicis is paralyzed, the patient uses flexor pollicis longus to hold the book and prevent it from slipping out of the hand. This results in flexion of the thumb— 'Froment's sign' and a positive test (Fig. 7.24).

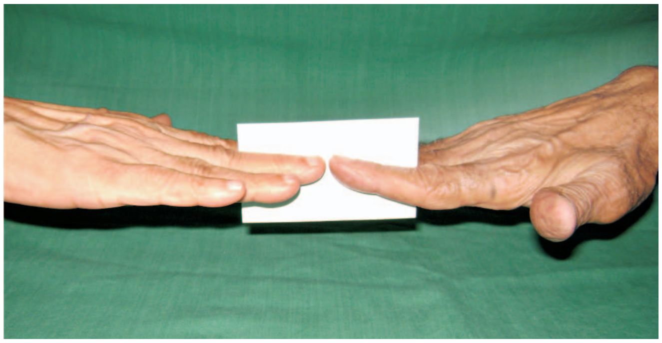


Figure 7.23

Card test being performed. Note that both the examiner and the subject to be examined are using the same grip, i.e. interdigital clasp.

Igawa's Sign

This is a quick test to assess the interossei. The patient is made to place the hand on the table and asked to raise the middle finger and move it side to side. If the interossei are paralyzed, he will not be not able to do so (Fig. 7.25).

Tinel's Sign

This sign was described by Jules Tinel, a French physician (1879–1952). The test is performed by percussion of the nerve along its course from distal to proximal there by causing stimulation. At the site where the nerve is irritable, patient experiences pins and needles/ tingling down the course of the nerve. It is a useful sign after nerve repair, when done at intervals and the findings are recorded.

Three types of responses are observed.

a. Strong response at the site of injury not progressing distally—indicates poor prognosis (recovery).
b. Fading response at the site of injury progressing distally—indicates good prognosis (recovery).
c. Persistent response at the site of injury as well as progressing distally—unpredictable prognosis (recovery).

Figure 7.24

Book test being performed and the Froment's sign. Note the flexion of the interphalangeal joint of the thumb (white arrow) which is brought about by the flexor pollicis longus.

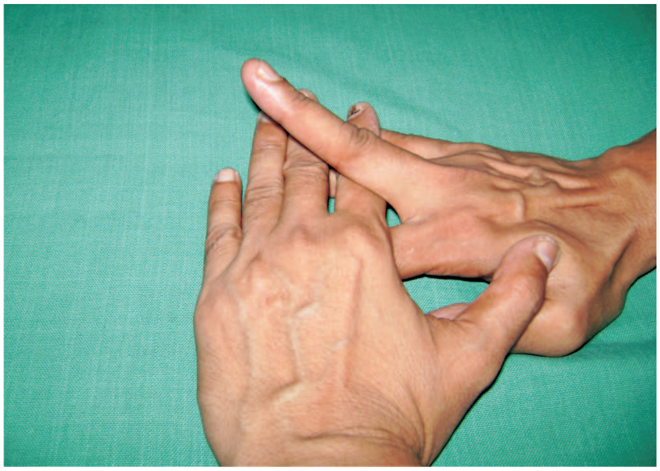


Figure 7.25

Igawa's test being performed. Note how the other fingers are stabilized and the middle finger is made to move sideways both medially and laterally, testing both palmar and dorsal interossei.

Table 7.1

Showing the deficits after nerve injury and the different tendon transfers

Nerve involved	Deformity	Muscles paralysed	Corrective procedures
Ulnar nerve high	Ulnar claw hand (Ulnar paradox)	FCU, FDP-III,IV, hypothenar muscles, interossei, medial two lumbricals, adductor pollicis	1. Zancolli's procedure 2. Extensor indicis/ extensor digiti minimi, tendon transfer
Ulnar nerve low	Ulnar claw hand	Hypothenar muscles, interossei, medial two lumbricals, adductor pollicis	1. Zancolli's procedure 2. Extensor indicis/ extensor digiti minimi, tendon transfer
Median nerve high	Ape thumb and pointing index	All the forearm and finger flexors except FCU and FDP to ring and little fingers, thenar muscles and lateral two lumbricals	1. Extensor indicis for opponens plasty 2. Lateral two profundi attached to medial two profundi which are supplied by ulnar nerve for combined action
Median nerve low	Ape thumb	Thenar muscles and lateral two lumbricals	1. Opponens plasty using extensor indicis
Radial nerve	Wrist drop	All the extensor muscles of the forearm, supinator and brachioradialis	1. Tendon transfer, Zachary's modification of Jones procedure. FCU to Ext. Digitorum, PL to EPL Pronator teres to ECRL and ECRB 2. FDS of middle and ring to ECRL/ECRB and ED; PL to EPL can also be used
Posterior interosseus nerve	Finger drop	All the extensor muscles of the forearm except, ECRL, ECRB, brachioradialis and supinator	1. Tendon transfer using brachioradialis for Ext. Digitorum, PL to EPL
Lateral popliteal nerve	Foot drop	Muscles of the anterolateral compartment of the leg	1. Tibialis posterior tendon to III cuneiform or III metatarsal brought through circumtibial/ interosseous route to the dorsum of the foot

Flexor carpi ulnaris—FCU; Flexor carpi radialis—FCR; Flexor digitorum profundus—FDP; Flexor digitorum superficialis—FDS; Palmaris longus—PL; Extensor pollicis longus—EPL; Extensor carpi radialis longus—ECRL; Extensor carpi radialis brevis—ECRB

Thenar group of muscles: Abductor pollicis brevis, Flexor pollicis brevis, Opponens pollicis.

Hypothenar group of muscles: Abductor digiti minimi, Flexor digit minimi, Opponens digit minimi.

Note: *The Tinel's sign does not predict the quality or quantity of regeneration. It also does not predict the return of function. It only suggests the type of nerve regeneration, i.e. whether the regeneration is proceeding in an orderly manner or not. Functional recovery is dependent on several other factors such as status of the muscle, status of the joint, presence of associated tendon injury, age of the patient, type of the nerve, etc.*

Revision Questions

Q. Describe the structure of a peripheral nerve.

Q. What are the clinical deficits that arise after a peripheral nerve injury?

Q. Classify nerve injuries.

Q. Discuss the following
- a. Neuropraxia
- b. Axonotmesis
- c. Neurotmesis

Q. What is Wallerian degeneration?

Q. What is electrodiagnosis?

Q. Write notes on:
- a. Rheobase
- b. Chronaxie
- c. Nerve conduction velocity
- d. Strength duration curve

Q. Write notes on:
- a. Claw hand
- b. Ulnar paradox
- c. Simian hand
- d. Ape thumb deformity
- e. Wrist drop and finger drop
- f. Foot drop
- g. Flail foot

Q. Write notes on:
- a. Card test
- b. Book test
- c. Pen test
- d. Igawa's sign
- e. High stepping gait

Q. Discuss the methods of repair of a peripheral nerve.

Q. Discuss nerve grafting.

Essay Questions

Q. Discuss the pathoanatomy, clinical features, diagnosis and management of a peripheral nerve injury.

Q. Discuss the response of a nerve to injury. Describe in detail the clinical features, diagnosis and management of a case of ulnar nerve injury. (The same question can be applied for other peripheral nerves also.)

Q. What is tendon transfer? Discuss the tendon transfers for:
a. Claw hand
b. Wrist drop
c. Median nerve injury
d. Foot drop

Q. Classify the reconstructive procedures designed for claw hand correction. Discuss the merits and demerits of the same.

BRACHIAL PLEXUS

FORMATION OF BRACHIAL PLEXUS

Brachial plexus is formed by the union of anterior rami of C5, C6, C7, C8 and T1. When C5 receives contribution from C4 it is known as pre-fixed and when T1 receives contribution from T2 it is known as post-fixed. The plexus comprises of roots, trunks, divisions, cords and branches (Figs 7.26 and 7.27).

C5 and C6 roots unite to form the upper trunk, C7 root alone continues as middle trunk, C8 and T1 roots unite to form the lower trunk. Each trunk divides into an anterior division and a posterior division. The anterior division of the upper and the middle trunk unite to form the lateral cord, the anterior division of the lower

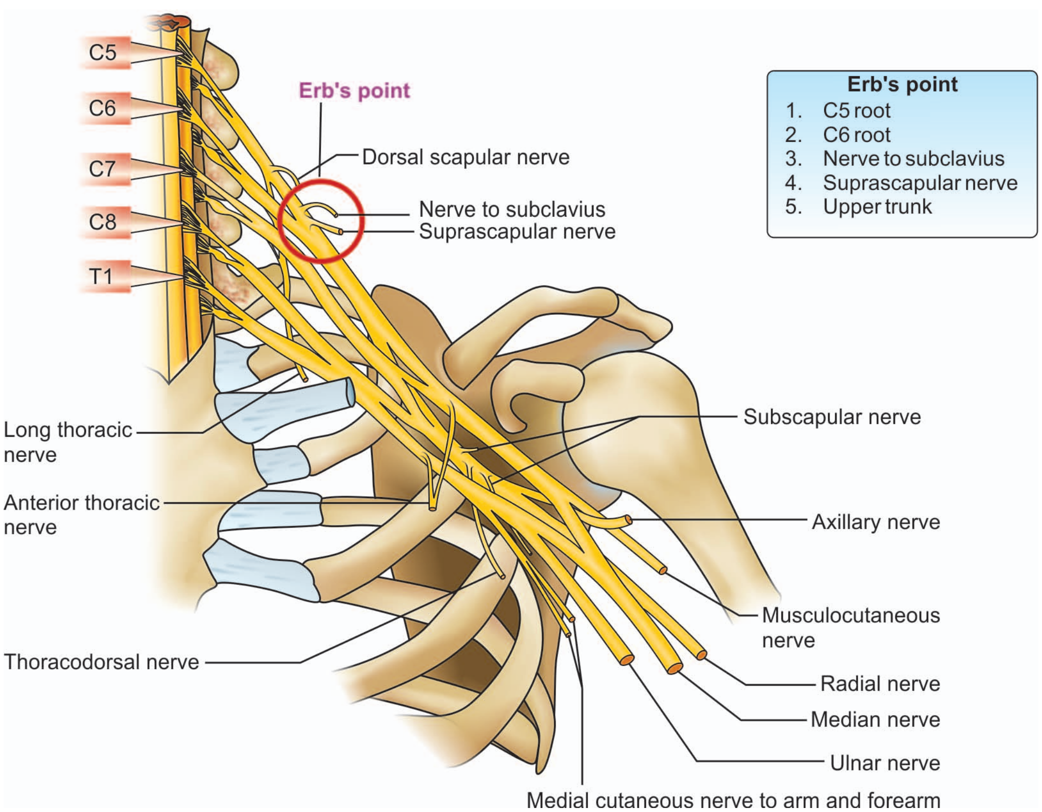


Figure 7.26

The drawing of the brachial plexus. C5,C6 roots join to form the upper trunk, C7 root continues as middle trunk and C8, T1 roots join to form the lower trunk. The circled area in red shows the Erb's point, a point of origin of five nerves.

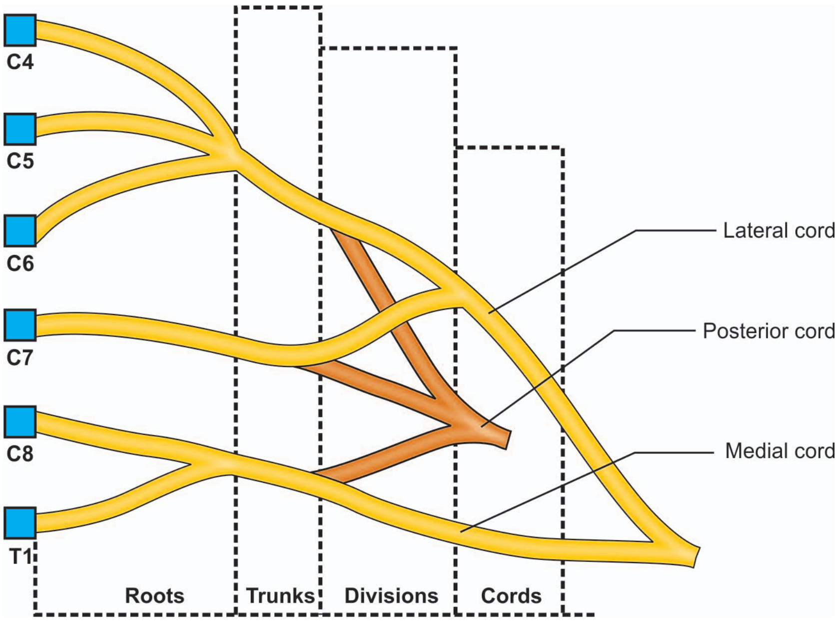


Figure 7.27

Brachial plexus with roots, trunks, divisions and cords. The plexus shown here is prefixed with contribution from C4.

trunk continues as medial cord and posterior divisions of all the trunks unite to form the posterior cord. The branches of the cord are as follows:

Lateral cord

a. Lateral pectoral
b. Musculocutaneous
c. Lateral root of the median nerve

Medial cord

a. Medial cutaneous nerve of the forearm
b. Medial cutaneous nerve of the arm
c. Medial pectoral
d. Medial root of the median nerve
e. Ulnar nerve

Posterior cord

a. Upper subscapular
b. Lower subscapular
c. Thoracodorsal
d. Radial
e. Axillary

Nerves arising from the roots

a. Long thoracic nerve—form C5, C6, C7 roots
b. Dorsal scapular nerve—from C5

Nerve arising from the upper trunk

a. Suprascapular nerve
b. Nerve to subclavius

INJURIES OF BRACHIAL PLEXUS

Brachial plexus can get injured either by direct penetrating wounds, e.g. assault, bullet and missile injuries or by traction force e.g. vehicular accidents, birth injuries in which the angle between the shoulder and the neck widens (increases).

A thorough knowledge of anatomy is essential for an accurate diagnosis with reference to the level of lesion. The lesion can be at the level of the roots, trunks, divisions or cords. The functional loss is directly related to the paralysis of the corresponding nerves which take origin from these. Examination of various muscles helps in arriving at a diagnosis.

Further, it is necessary to distinguish between pre and postganglionic type of injury. The prognosis in preganglionic type is poor as repair is difficult. Prognosis in postganglionic type is better as repair is possible. Absence of 'Horner's syndrome' and cutaneous axon reflex indicate that the lesion is postganglionic.

Signs of Preganglionic Lesions

a. Intractable burning pain in a sensationless, paralyzed extremity.
b. Presence of spinal fractures.
c. *Horner's syndrome:* It is characterized by ptosis, enophthalmos, *miosis* and anhydrosis.
   (Other names for Horner's syndrome are Bernard-Horner syndrome, Claude Bernard-Horner syndrome or oculosympathetic palsy)
d. *Cutaneous axon reflex:* This reflex is elicited by inducing irritation by placing a drop of histamine over the skin along the distribution of the nerve to be examined and scratching the site (may also be done by injecting histamine intradermally). Normal reaction is vasodilation followed by wheal and a flare (triple response). This reflex is absent in postganglionic lesions where the response is only a wheal. No flare response is seen.
e. Posterior cervical electromyography may show denervation potentials in the posterior cervical paraspinal muscles indicating that the lesion is preganglionic.
f. Presence of long tract signs in the lower limbs indicating the involvement of the spinal cord and the proximal nature of the injury.
g. CT myelography/MRI may show pseudomeningocele indicating root avulsion and preganglionic lesion. These investigations are unreliable during the early phase as dural tears can also give a false picture of pseudomeningocele.

Upper Plexus Injury—Erb's Paralysis (Fig. 7.28)

It is characterized by involvement of C5, C6 nerve roots. (C7 involvement may or may not be present).

Functional loss results in a typical attitude of the upper limb. The limb remains by the side of the trunk with shoulder in adduction and internal rotation, elbow in extension, forearm in pronation and wrist in flexion and ulnar deviation. This is because of the paralysis of abductors, namely the deltoid and supraspinatus and external rotators, namely infraspinatus and teres minor at the shoulder; paralysis of flexors, namely the biceps, brachialis and the brachioradialis at the elbow; paralysis of the supinator at the forearm.

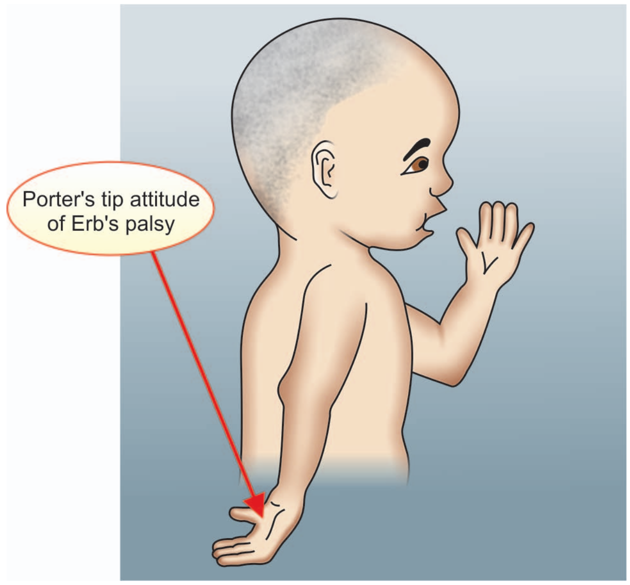


Figure 7.28
Characteristic attitude of 'the Porter's tip hand' of Erb's palsy.

Because the limb hangs by the side of the body and has an attitude that resembles the attitude of *a porter waiting for a tip,* it is known as *"the porter's tip hand"*.

Sensory loss over the deltoid, lateral aspect of the forearm and hand is seen.

Paralysis of the serratus anterior supplied by long thoracic nerve, rhomboids and levator scapulae muscle supplied by dorsal scapular nerve, indicates that the lesion is proximal to the origin of these nerves (these nerves take origin from the roots, before the plexus is formed) and may be preganglionic.

Lower Plexus Injury—Klumpke's Paralysis

It is characterized by involvement of C8 T1 nerve roots. C7 involvement may or may not be present.

Functional loss/deficit is both sensory and motor involving C8 T1 roots. This is very characteristic. Hence, there is paralysis of intrinsic muscles of the hand along with wrist and finger flexors. The sensory deficit is on the medial aspect of the hand, forearm and arm.

Whole Plexus Injury

It is characterized by complete flaccid paralysis of the entire upper limb and considered as one of the most serious injuries as chance of recovery is very poor.

Injuries of the Trunks

Injuries of the upper trunk manifest with similar deficits as their rami (branches). But, the long thoracic and dorsal scapular nerve escape paralysis.

Injuries of the lower trunk manifest with similar deficits as their rami (branches). But, there is no sympathetic involvement and Horner's syndrome.

Injuries of the Divisions

Injuries of the divisions of the plexus is extremely rare and it is difficult to distinguish from trunk and cord injuries.

Injuries of the Cords

Injuries of the cords manifest with paralysis of the nerves which take origin from the respective cords. Paralysis of the muscles supplied by these nerves and sensory loss in the corresponding autonomous zones are the diagnostic features.

Management

Methods available for the management are:

Nonoperative

Employed only after confirming that the injury is I or II degree according to Sunderland classification. It should never be employed in more severe disruptions and in open injuries of the brachial plexus.

Appropriate splints are given as per the deficit to give rest to the paralyzed part and prevent contractures, e.g. shoulder abduction splint, spica cast, etc.

Drug therapy is given to reduce edema and increase vascularity. Electrical stimulation and exercise therapy is given during the phase of recovery.

Operative

Immediate surgery is indicated only in open injuries of the plexus. Otherwise, the surgeries for brachial plexus injury, are always well planned and done at a later date.

Surgical Procedures

i. Repair—It is done when there is partial or complete neurotmesis.
ii. Interfasciculus nerve grafting—It is done when there is loss of nerve tissue.
iii. Neurolysis ???—This is done when there is scarring around the plexus (contradiction exists with neurolysis. The role of neurolysis is questionable.)
iv. Neurotization and nerve transfer—This is done in root avulsion where repair is not possible. It is a highly specialized procedure.

Some of the recommendations are as follows:

Neurotization

Avulsion of C5 C6 roots: Spinal accessory nerve to suprascapular nerve; two or three intercostals nerves to musculocutaneous nerve.

Nerve grafting:

If C5 root is available, it is grafted to lateral cord to provide elbow flexion, finger flexion and sensation along the radial side of the hand.

If C5 C6 roots are available, they are grafted to posterior and lateral cords.

Results of neurotization will be evident only after a period of 2 to 3 years. Good postoperative care, physiotherapy and regular follow-up is essential for successful outcome.

Reconstructive procedures: Procedures such as tendon transfers with available tendons, can restore some useful function. Corrective osteotomies to correct rotational deformities, if any, are indicated in cases where recovery is poor.

Note: *The treatment of brachial plexus injury is challenging. Highly specialized care is necessary for useful functional recovery. Detailed description is beyond the purview of this book and hence not described. The description limits itself to basic principles to be followed in the treatment.*

Revision Questions

Q. Erb's paralysis.
Q. Klumpke's paralysis.
Q. Whole arm paralysis.
Q. Erb's point.

Essay Question

Q. Discuss the pathoanatomy of the brachial plexus. Describe in detail the mechanism of injury, clinical features, diagnosis and management of brachial plexus injury.

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8 Bone and Joint Infections

- *Introduction*
- *Etiopathogenesis of nonspecific bone and joint infections (osteomyelitis and pyogenic arthritis)*
- *Clinical features and diagnosis*
- *Treatment and outcome of treatment*
- *Complications and treatment of complications*
- *Etiopathogenesis of specific bone and joint infections: bone and joint tuberculosis, mycotic infection, syphilitic infection, etc.*
- *Clinical features and diagnosis of specific bone and joint infections*
- *Treatment and outcome of treatment*

INTRODUCTION

When medical fraternity got convinced that microbes were responsible for bone and joint infections, a new era began. This changed the scenario in the mid-19th century and 20th century witnessed the discovery of several antibiotics starting from penicillin and sulfa to the more recent, cephalosporins, imipenem, etc. In spite of this, even today, bone and joint infections pose a challenge to an orthopedic surgeon. Effective control of infection is many a time not possible, especially when bacterial resistance is encountered or if the patient is immunocompromised.

Osteomyelitis

The word osteomyelitis, is derived from Osteon (bone), myelo (marrow), itis (inflammation). It is said to be coined by Nelaton in the year 1834. ***Hence osteomyelitis is defined as inflammation of the bone and the bone marrow.*** This word osteomyelitis has become synonymous with infection of the bone and the bone marrow because, most of the time, it is the infection that causes this inflammation. Other causes for inflammation such as thermal and chemical burns, mechanical trauma and radiation exist in less than 1% of cases and in such cases secondary bacterial infection invariably follows.

Route of Entry of Microorganism

Endogenous: In this type, the organism enters the bone from an infective focus existing elsewhere in the body. Two modes of spread have been identified.

i. Local: When the infective focus is present in the vicinity of the bone, the infection spreads to the bone by direct extension, e.g. abscess in the soft tissue.
ii. Distant: When the infective focus is a distant one, e.g. a septic tooth (caries tooth), septic tonsil, chronic otitis media, etc. the infection spreads through blood stream. This is known as hematogenous spread (most common).

Exogenous: In this type, a break in the soft tissue should occur. This allows direct entry of the organisms from the environment, e.g. penetrating wound, open fracture, etc.

Specific Osteomyelitis

When the bone is infected with those organisms which cause specific features of a disease, e.g. tuberculosis,

syphilis, mycotic infection, etc. it is known as specific osteomyelitis. The terminologies such as tuberculosis of the bone, mycotic infection of the bone, etc. are also used to describe the pathology.

Nonspecific Osteomyelitis

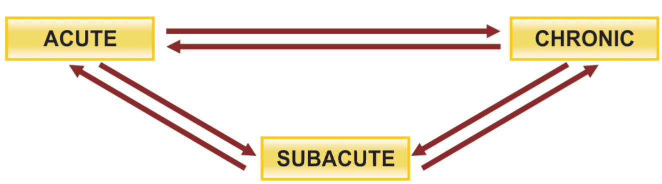
When the bone is infected with those microorganisms which produce general features of infection in the bone (without specific features), it is known as nonspecific osteomyelitis.

NONSPECIFIC INFECTION

Acute Hematogenous Osteomyelitis

This is a progressive infection of the bone and the bone marrow resulting in destruction.

Simultaneously, the body reacts by extruding the dead and devitalized tissue and forming new bone. This reaction is considered as a part of natural process of healing. Many a time, it is not possible to eradicate the focus of infection completely and a persistent discharging sinus remains suggesting chronicity. Thus, *the infection starts as an acute process and progresses to remain as subacute or chronic when total eradication of infection is not achieved.* Reactivation of this persistent subacute or chronic focus of infection into acute form is also possible.



Etiopathogenesis

Predisposing factors

Age: Common in children. Less common in adolescents and adults (adult risk factors are immunocompromised status, diabetics, drug addiction).

Sex: M:F ratio is 5:1.

Trauma: Trauma may cause a hematoma which may get secondarily infected and result in osteomyelitis.

Location: Metaphysis of a long bone.

Presence of septic focus: A septic tonsil, septic tooth, chronic otitis media, enteric fever septicemia, etc.

Poor nutritional status: General debility and malnutrition.

Organisms responsible: Staphylococcus aureus, being the most virulent human pathogen, is the most common organism that causes acute hematogenous osteomyelitis (>95% of cases). Other organisms include *H. influenzae, Streptococcus, E. coli, Proteus,* etc. (< 5% of cases). *Salmonella typhi* infection is more common among patients with sickle cell anemia.

Pathology

The pathology begins with *entrapment of infective emboli* in the metaphysis of a long bone causing occlusion of the vessels. It is said to get entrapped in this region because of the following reasons:

- Metaphysis is more vascular.
- Most metaphyseal arteries are end arteries.
- Vessels show a hairpin bend.
- Vascular lumen is of a narrower caliber.
- Relative lack of phagocytosis.

Thus, avascular necrosis occurs distal to the block. The dead tissue acts as a good media for the bacteria to multiply. At the same time, the body defense mechanisms try to overcome the infection. Hence, the ensuing antigen antibody reaction results in the *formation of an exudate.* At this stage, if the body defense mechanisms overcome the infection, a small exudate forms which subsequently gets absorbed. The clinical symptoms subside and no further destruction takes place. But, if the organism is of high virulence and body defense mechanisms fail to overcome the infection, the process of destruction continues and a large exudate, a bone abscess forms. This large exudate, exerts pressure on the surrounding vessels and causes *compression*, *further occlusion, ischemia and necrosis.* Thus, a vicious cycle sets in and all the systemic signs of an acute infection develop and persist.

The bone abscess thus formed has to find its way out. Its path is blocked proximally, by the epiphysis and the growth plate. Its spread into the joint is prevented by the attachment of capsule at the epiphysiometaphyseal region. So, the *only way it can flow, is down the medullary canal*. It can *also come out and lie in the subperiosteal region* through the normal openings in the bone, the Haversian and the Volkmann's canals as well as the openings for the nutrient vessels. Thus, there develops a *subperiosteal and intramedullary abscess.* This subperiosteal abscess ultimately finds its wayout by *breaking the fascia, subcutaneous tissue and the skin*

and a sinus of chronic osteomyelitis develops. The whole process takes about 14–21 days and upon development of this draining sinus, all the acute signs of infection subside.

That *part of the bone between the intramedullary and the subperiosteal abscesses, loses blood supply* (the subperiosteal abscess cuts off the periosteal blood supply and the intramedullary abscess cuts off the endosteal blood supply). This *results in the formation of a sequestrum.* Smaller sequestra, are absorbed by the body and the larger sequestra, which cannot be absorbed are thrown out by the body through the sinus (Hence, the classical history of a discharging sinus with bony spicules, is obtained in chronic osteomyelitis). Larger sequestra which can neither be absorbed nor be thrown out, remain and contribute for a persistent discharging sinus with sprouting granulation tissue at its mouth (Fig. 8.2).

At the same time, *the periosteum forms* new bone from its deeper cambial layer. This is visible radiologically as a layer of new bone formation and is known as *involucrum.*

Sequestrum is defined as 'a dead bone in situ' or a dead bone within a living bone or a dead bone surrounded by granlulation tissue within a living bone.

Involucrum is defined as a reactive periosteal new bone formation seen in osteomyelitis (Fig. 8.1A).

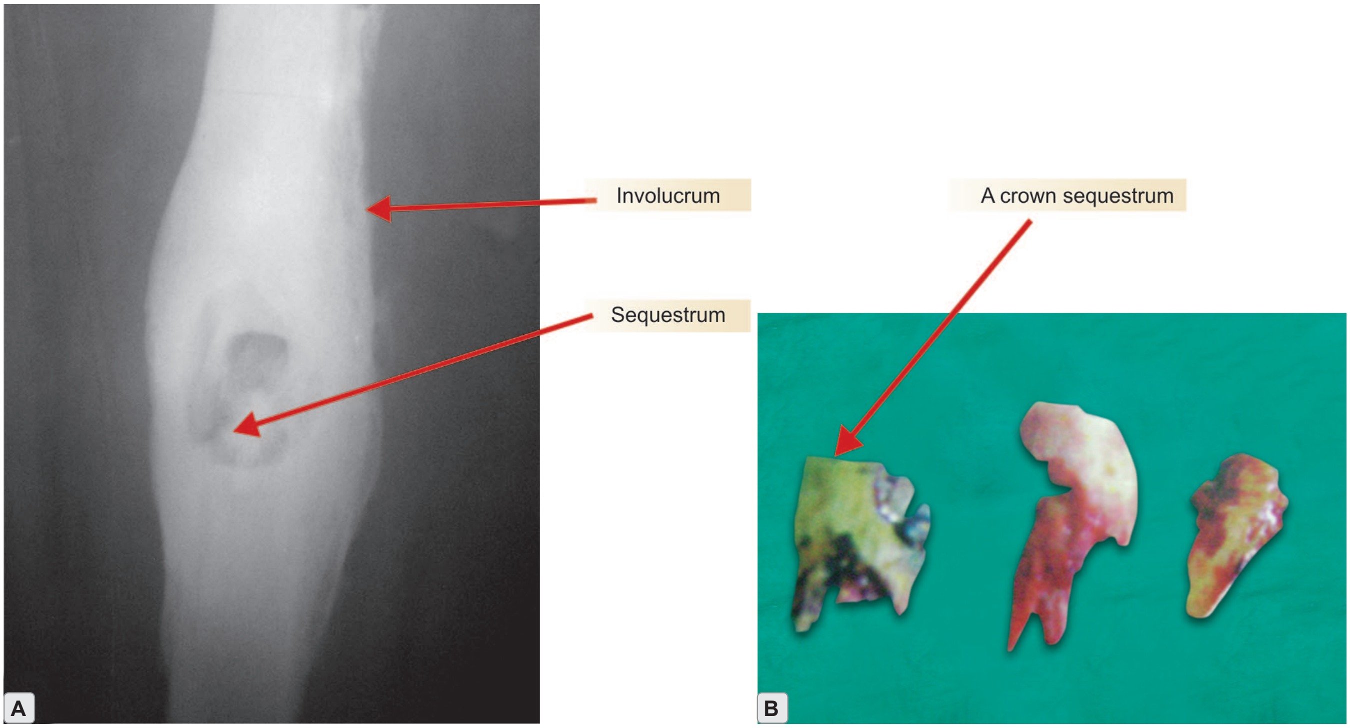
A sequestrum is identified by its dusky naked eye appearance. The margins of the sequestrum are always irregular. It is always found in a cavity and is surrounded by unhealthy granulation tissue.

Sequestrum is identified radiologically by:

i. Its increased density than the surrounding normal bone (Fig. 8.1A).
ii. A parasequestral clear zone of unhealthy granulation tissue which casts a radiolucent shadow.
iii. Its irregular margins.
iv. It is being surrounded by involucrum (at times, not always).

Types of Sequestrum (Fig. 8.1B)

Ivory—seen in syphilitic osteomyelitis
Black—in fungal osteomyelitis
Sandy or rice grain—tuberculosis
Diaphyseal—large tubular, seen in children
Crown—in amputation
Ring—infected pin track
Feathery—tuberculosis involving the ribs



Figures 8.1A and B

(A) Sequestrum and the involucrum. (B) Types of sequestrum.

Clinical Features

- Presence of focus of infection elsewhere in the body.
- Evidence of local trauma.
- Abrupt onset of high grade fever.
- Signs of systemic toxicity.
- Swollen, tender, edematous, limb with refusal to move (pseudoparalysis).
- Effusion may be present in neighboring joints.

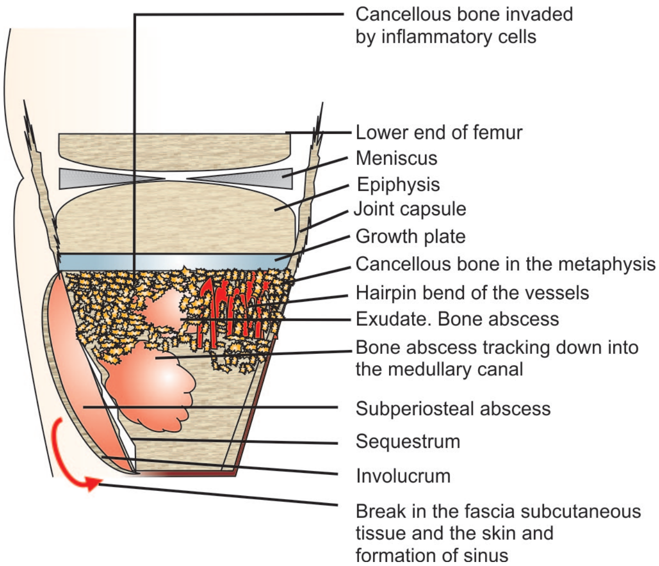


Figure 8.2

Diagrammatic representation of the pathology and progress of acute hematogenous osteomyelitis.

Investigations

Blood investigations

Hb%—may be low.

TC—leukocytosis, DC—increase in neutrophils, ESR—elevated.

CRP—elevated levels.

Blood culture—positive for the organism.

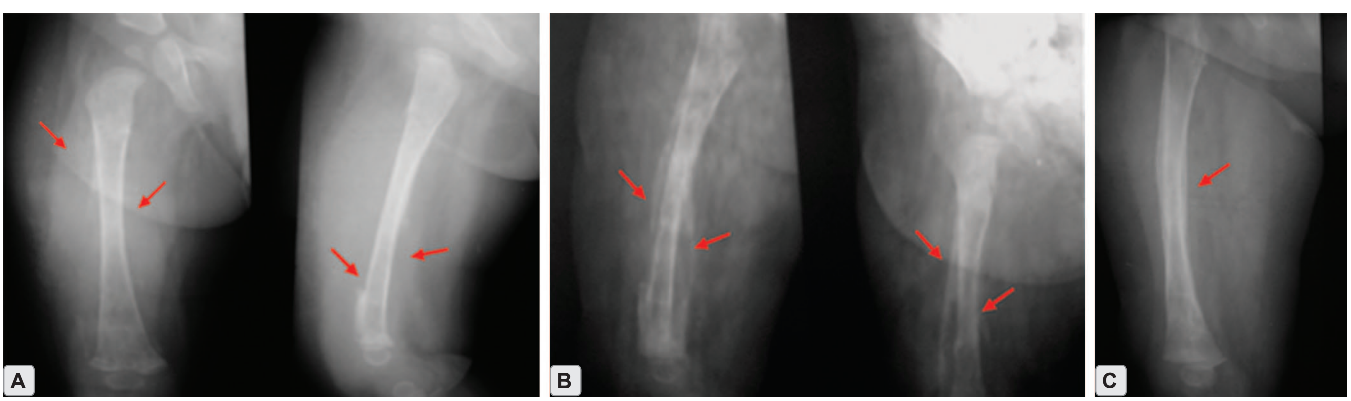
Other Investigations

X-ray—does not show any signs of infection early in the disease. The first positive radiological sign is periosteal elevation which takes 2–3 weeks (Figs 8.3A to C). Hence, X-ray is not of use in early diagnosis (Fig. 8.4A). Sequestrum and involucrum are seen radiologically around 4–6 weeks, only when the infection establishes and becomes chronic.

MRI (Fig. 8.4B), bone scan and CT are of use in the order of preference. These help to confirm the clinical diagnosis as well as to delineate the extent of inflammation.

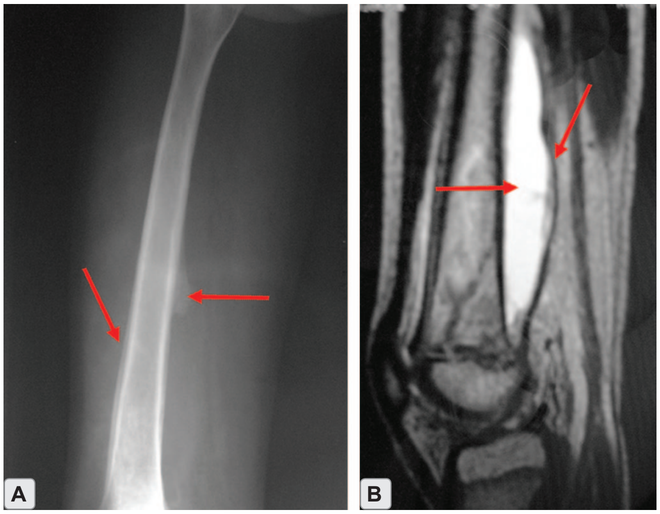
Treatment

Acute osteomyelitis: Aim of the treatment is to *eradicate the infection* and prevent progression to chronic osteomyelitis. So, once the diagnosis is made, the treatment should be carried out *on an emergency basis.* As time lapses, infection spreads. This poses difficulty in achieving total eradication. The systematic approach to be followed is as follows:



Figures 8.3A to C

Serial radiographs of different stages of osteomyelitis in children, showing the outcome following drainage of pus, protection with a splint and administering adequate antibiotic therapy: (A) The first positive radiological sign is the periosteal reaction which takes about 14 days to 21 days to develop. Since periosteum is loosely attached in children, the spread of infection is extensive along the diaphysis. So is the periosteal reaction; (B) Radiograph taken at 6 weeks showing a large diaphyseal sequestrum, pronounced periosteal reaction and formation of involucrum following drainage of pus; (C) Complete resolution at 3 months following drainage of pus and adequate antibiotic therapy. Note that no sequestrectomy has been done.



Figures 8.4A and B

(A) Radiograph of the femur 3 weeks after the onset of the disease, showing periosteal new bone formation. (B) MRI done at the same time shows that there is collection of fluid around the femur. This collection of fluid, lifts the periosteum. Hence, the new bone formation occurring in the cambial layer of periosteum becomes visible (Involucrum).

a. *Antibiotics: Broad spectrum antibiotics* are to be administered immediately after drawing the sample for blood culture.
b. *Drainage of pus:* The pus inside the bone (bone abscess) has to be drained. The *drainage* procedure is known as *decompression.* This can be accomplished by making multiple drill holes or cutting a large window in the involved bone, for the drainage of pus. The pus which is under tension is drained out by this procedure. Hence, the name decompression. It is done under broad spectrum antibiotic cover and later followed up with adequate specific antibiotic therapy depending on culture and sensitivity report.
c. *Control of infection:* Locally the *method of continuous irrigation and suction,* i.e. infusing fluid containing appropriate antibiotics can be employed to achieve the eradication of infection.
d. *Immobilization:* The limb is kept immobilized and protected with the help of external splint. Long-term antibiotic therapy is necessary for total eradication. The potential hazards of long-term therapy have to be considered and observed during the period.
   Note: *Since Staphylococcus aureus is the most common pathogen for acute hematogenous osteomyelitis, Penicillin group of drugs are the best to begin with. Later on, according to the blood culture or pus culture reports, appropriate drugs can be administered.*
e. *Eradication of infection:* Administration of prolonged, adequate and appropriate antibiotic therapy eradicates the focus of infection (Figs 8.5A to F).

Complications

General

i. Septicemia, pyemia, multiple pyemic abscesses, Death.
ii. Amyloidosis.

Local

i. Chronic/subacute osteomyelitis/pyogenic arthritis.
ii. Pathological fracture.
iii. Limb length discrepancy.
iv. Squamous cell carcinoma of the sinus tract.
v. Deformity.

Chronic Osteomyelitis

When the infection is established in the bone, it results in chronic osteomyelitis.

Clinical features

- Persistent discharging sinus fixed to the bone
- Bony thickening
- Bony irregularity
- Bony tenderness may be present
- Deformity may be present
- Limb length discrepancy may be present

Radiological features

- Bony irregularity
- Increased density
- Presence of cavities
- Presence of sequestrum
- Presence of involucrum

Investigations

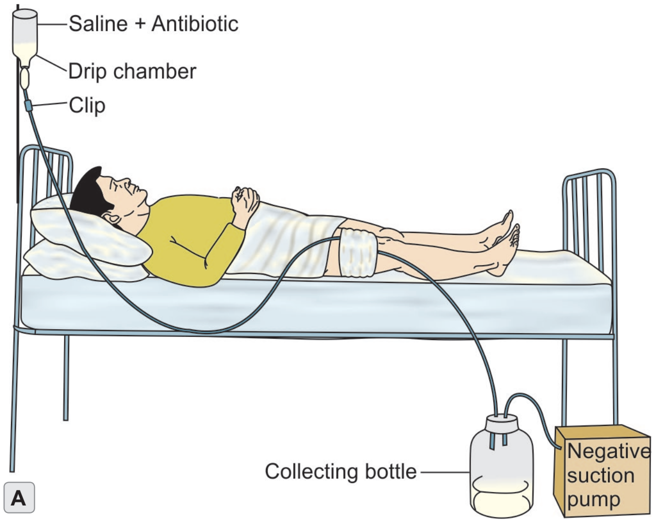
a. Routine blood investigations. Hb%, TC, DC, ESR, CRP, etc.
b. Pus for culture sensitivity.
c. X-ray of the involved bone shows typical features. (Ref: X-ray—in acute osteomyelitis).

Treatment

- Adequate and specific long-term antibiotic therapy.
- Sequestrectomy and saucerization:
  – Removal of the sequestrum is done only when the sequestrum has fully separated as confirmed by a parasequestral clear zone and

Figures 8.5A to F

Natural course of acute osteomyelitis, presented late for treatment: (A) Though there was pus inside the bone X-ray did not show any abnormality; (B) Three weeks after decompression and adequate drainage of pus; (C and D) Sequestrum formation without any involucrum, at 12 and 18 weeks respectively; (E) Defective nonunion after sequestrectomy at 24 weeks. Note the intact fibula; (F) Successful results after sliding bone graft and cancellous grafting along with attempted tibialization of fibula. Procedure was done after complete *eradication of infection*.



Figures 8.6A and B
Application of continuous irrigation and suction in an attempt to achieve control of infection.

presence of a healthy involucrum. The deep cavity which contains the sequestrum is made shallower in the form of a saucer to facilitate the drainage by lymphatics and venules.

- Continuous irrigation and suction system may be employed (Figs 8.6A and B).

Subacute Osteomyelitis

It is a distinct form of bone infection where organisms present in the bone, do not cause classical symptoms of either acute or chronic osteomyelitis. The only symptoms present may be local pain and swelling. This can occur either primarily, when the virulence of the organism is low or the host resistance is high or secondarily, when acute osteomyelitis turns to subacute after treatment.

Brodie's Abscess

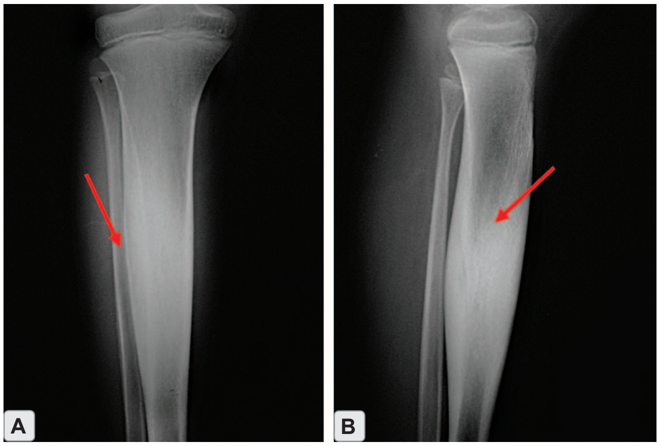
It was described by Sir Benjamin Brodie, a surgeon in St George's Hospital, London, in 1832.

He found a subacute infection in a specimen of a limb which he had amputated for intractable pain. He observed a cavity filled with dark colored thick pus with walls made up of hard white bone.

Age of onset—5–15 yr

Sex—M:F ratio 2:1

Site—Diaphysis is common. Metaphysis is the next common site. Rare in the epiphysis.



Figures 8.7A and B
Brodie's abscess in the diaphysis of tibia. The only complaint the child had was low grade pain. Limp, local signs of acute inflammation and constitutional symptoms were absent.

Clinical features: Presents with mild to moderate pain. A hard swelling may be appreciated.

Radiological features: The disease presents with variable features. Classically, a cystic lesion surrounded by sclerotic bone is the feature. But, in some instances onion skin periosteal bone resembling Ewing's, cortical hyperostosis resembling osteoid osteoma, etc. are observed (Figs 8.7A and B).

Treatment

Once the diagnosis is established long-term broad spectrum antibiotic therapy is started.

Surgery for draining the cavity under antibiotic cover is considered only when nonoperative treatment fails. (bacteriological culture sensitivity report from the sample obtained from the cavity has to be considered when the abscess is drained.)

Sclerosing Osteomyelitis of Garre

It is a nonsuppurative ossifying periostitis which occurs as a response to low grade infection and ensuing irritation. Radiograph shows sclerosis all round the bone.

Bone scan and PET scan may be useful in the diagnosis. Treatment consists of adequate antibiotic therapy or when feasible and indicated, excision of the focus.

Revision Questions

Q. Define osteomyelitis.

Q. What are the organisms responsible for acute hematogenous osteomyelitis?

Q. Discuss the mode of spread of infection to the bone in osteomyelitis.

Q. Discuss the pathogenesis of chronic osteomyelitis.

Q. Define sequestrum.

Q. Define involucrum.

Q. Write notes on

a. Decompression.

b. Sequestrectomy and saucerization.

c. Brodie's abscess.

d. Sclerosing osteomyelitis of Garre.

Q. What are the complications of acute hematogenous osteomyelitis?

Essay Questions

Q. Discuss the etiopathogenesis, diagnosis and management of acute hematogenous osteomyelitis. Enumerate the complications and discuss in detail the management.

Q. Discuss the pathogenesis of chronic osteomyelitis, its clinical features, diagnosis, complications and management.

Acute Suppurative Arthritis

This entity is known by various names such as acute pyogenic arthritis, acute septic arthritis, acute pyarthrosis and acute infective arthritis. All of these refer to presence of pus in the joint secondary to infection of the joint.

Age of onset—common in infants and children; can occur at any age.

Sex—M:F ratio 5:1

Site—Any joint. Common in hip and knee.

Mode of Spread

- Exogenous, e.g. penetrating injury.
- Endogenous, e.g. hematogenous from a distant focus; local extension from the vicinity.

Organisms

Staphylococcus aureus in 90% of the cases, *Streptococcus, H. influenzae, E. coli, Proteus*, etc. are responsible in remaining 10% of the cases.

Focus of infection—originates from the synovium or from the bone then spreads.

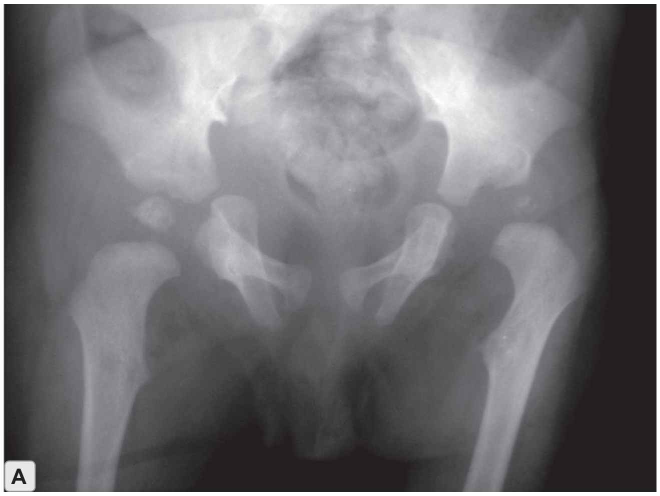
Pathology

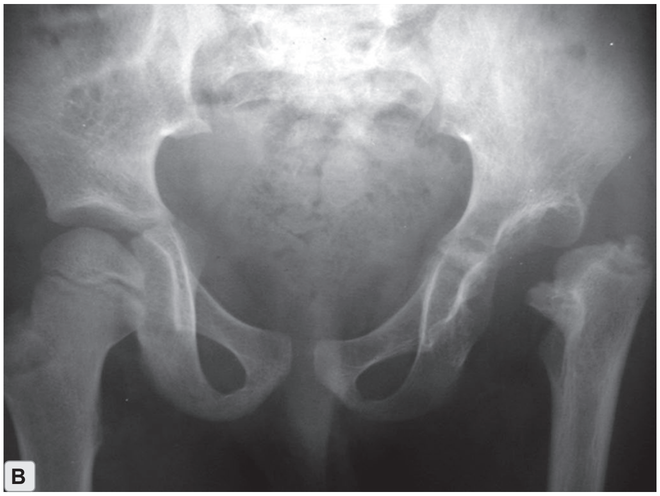
Once the infection gets into the joint, there is a seropurulent exudate formation. The articular cartilage gets destroyed very rapidly due to the enzymatic degradation by enzymes released from the bacteria as well as from the inflamed synovium and inflammatory cells. The subchondral bone gets exposed and later gets eroded. If the pus is not drained surgically, a sinus develops.

Sequel depend on the speed at which the infection is controlled. If the infection is controlled before the destruction of the cartilage, the joint becomes normal. No residual deformity is seen. After some destruction of cartilage, the joint goes in for fibrous ankylosis. After considerable destruction of cartilage, the joint goes in for bony ankylosis. If the infection takes its natural course, bony ankylosis with deformity is the end result.

Diagnosis

Diagnosis is made mainly on clinical grounds. It is not difficult because the patient presents with all the signs of acute infection, i.e. calor, rubor, dolor, tumor with painful limitation of joint movements. Signs of systemic toxicity such as high grade fever with delirium may also be present. Investigations such as TC, DC, ESR, CRP, ultrasound, X-ray, MRI are useful and should be done as indicated.





Figures 8.8A and B

(A) The sequel of a Tom Smith's arthritis at 2 years. At the age of three months the infant had presented with the disease within 24 hours of onset of symptoms. The pus was drained under adequate antibiotic cover within 24 hours of presentation. Though treated aggressively and control of infection was achieved, note the hypoplastic change in the left femur and pathologic dislocation of the left hip joint; (B) An adolescent with sequel of Tom Smith's arthritis of infancy showing complete destruction of the femoral epiphysis and dislocation. This results in gross shortening and exaggerated movements in the hip. The hip is painless.

Treatment

Immediate: Emergency surgical drainage of pus and debris from the joint and under cover of systemic antibiotics is the treatment of choice. Continuous irrigation and suction can be employed for achieving good control of infection. Immobilization is essential till control of infection is achieved. This is followed by gradual mobilization as early as possible to avoid stiffness of the joint. Systemic antibiotics are used for a considerable period to get rid of any residual focus of infection depending on the organism isolated in culture and its antibiotic sensitivity.

Treatment of sequel: Arthrodesis is the treatment of choice for unsound ankylosis. In selected cases of hip and knee ankylosis with no reactivation of the disease, total joint arthroplasty can be considered. This procedure considerably reduces the morbidity.

Tom Smith's Arthritis of Infancy

This is a type of pyogenic arthritis occurring due to osteomyelitis of the upper femoral metaphysis. The focus of infection can also begin from the synovium. Since the capsular attachment is beyond the physis, the infection directly spreads into the joint causing pyogenic arthritis. The epiphysis invariably gets destroyed and the hip dislocates. This is an example for pathological dislocation.

Early and accurate diagnosis followed by prompt drainage of pus under antibiotic cover may reduce the morbidity. Delay causes severe destruction of the joint due to release of lysosomal enzymes. Increase in pressure within the joint, further disturbs the vascularity and promotes destruction (Figs 8.8A and B).

Revision Questions

Q. Define septic arthritis.

Q. Discuss the etiopathogenesis of acute septic arthritis.

Q. Discuss the treatment and sequel of acute pyogenic arthritis.

Essay Questions

Q. Discuss the etiopathogenesis, clinical features, diagnosis, management and complications of acute pyogenic arthritis.

Q. What is Tom Smith's arthritis? Discuss the diagnosis and management of Tom Smith's arthritis and its sequel.

SPECIFIC INFECTIONS

Tuberculosis of Bones and Joints

The disease tuberculosis is as ancient as mankind. Hippocrates during his period recorded this disease as phthisis or consumption. In Charaka and Sushrutha Samhitha in ancient India, it is identified as Yakshma. Studies on Egyptian mummies (2050 BC) have shown positive results for M. tuberculosis, M. africanum and M. bovis. Hippocrates was able to differentiate pyogenic abscesses from tubercular cold abscesses and to point out the fact that paraplegia in tuberculosis of the spine improved once the cold abscess pointed over the back (natural decompression).

Tuberculosis of bones and joints is always secondary to a primary focus elsewhere in the body. Many a time, this primary focus is not detectable. The bacilli, i.e. Mycobacteriae, enter the bone (dissemination occurs) through the bloodstream and get lodged in different areas. Thus, the lesions may be found in the synovium or in the bone. It is said that it takes almost 18–24 months for a primary lesion to cause bone and joint tuberculosis. Poor nutrition, poor sanitation and poor resistance are the predisposing factors for this infection. Age is not a bar for bone and joint tuberculosis which can occur at any age. But, when it occurs at a young age, the destruction of growing bones result in severe deformities. This is specially true in the spine.

The 3 stages of this disease that were identified in the pre-antitubercular drug therapy era are:

- I Early or stage of onset.
- II Florid or stage of active destruction.
- III Stage of repair or stage of healing or stage of ankylosis.
  (Was seen only in those who survived. More than 70% died from this disease).

The typical lesion of tuberculosis, is known as a tubercle which is histopathologically a granuloma. It consists of a central area of caseation necrosis, surrounded by epitheloid cells, macrophages, lymphocytes and Langhans' type of giant cells. It indicates that granuloma is a defensive response to bacterial invasion. This response, not only cordons off the bacteria but also allows the immune system to kill the bacteria. The T lymphocytes (CD4+) secrete cytokines (interferon gamma) which activate macrophages to destroy the engulfed bacteria. T lymphocytes (CD8+) also directly kills the infected cell.

Investigations

General:

- Hb%—low; TC—moderate increase; DC—predominantly lymphocytes; ESR-elevated.
- X-ray—always shows narrowing of the joint space as an earliest feature followed by destruction of the adjacent bones. Surrounding area of rarefaction is a striking feature. Soft tissue shadow of cold abscess may also be seen.
- CT, MRI—dilineate extent of soft tissue and bone involvement better than X-rays.
- Mantoux test—when positive, suggests that the patient has or has had tuberculosis/exposure to tuberculous bacilli.

  The test can be negative when there is no exposure to tuberculosis as well as in milliary tuberculosis.
- IgA and IgG—immunoglobulin assay may be useful as an additional evidence.

Diagnostic and confirmatory investigations:

- Tissue samples showing positive PCR (polymerase chain reaction).
- Smear showing presence of acid fast bacilli.
- Biopsy showing classical picture of a tuberculosis (a granuloma).

Treatment

Standard medical therapy in an adult comprises of using a combination of at least three of the following drugs.

- Streptomycin (0.75–1.0 G).
- Isoniazid—INH (200–300 mg).
- Rifampicin (300–450 mg).
- Ethambutol (800–1200 mg).
- Pyrazinamide (750–1250 mg).

Drugs are used as per body weight. Continuation of therapy for a minimum period of 18 months is absolutely necessary for cure. Daily regimen ensuring regular intake of drugs gives the best results.

Good sanitation and good nutrition plays a great role in the recovery.

DOTS trial is yet to prove its efficacy for skeletal tuberculosis and so are short-term therapies. It is the author's experience that reactivation of the lesion and manifestation as some other form of tuberculosis is common with DOTS and short-term antitubercular therapy in spite of taking drugs as per the schedule.

Role of surgery in active tuberculosis is to debride the tuberculous debris and repair the bone loss by adopting a stabilization procedure. It aims at rendering a subject totally disease free at the same time attempting to restore the functional capacity to the best possible extent. It also aims at preventing future complications.

Surgery is always done under cover of antitubercular drug therapy (ATT). The drug therapy is continued even after surgery as per schedule. Aim of surgery in a burnt-out case is reconstruction as well as deformity correction, thereby attempting to improve the function and cosmesis.

Injection streptomycin has the disadvantage of administration by intramuscular route and over a period of time the patient may become noncooperative. Ototoxicity is a common complication in elderly patients; hence, to be used with caution.

Essay Question

Q. Discuss the clinical features, diagnosis and drug therapy in bone and joint tuberculosis.

Tuberculosis of the Spine

Spinal tuberculosis is the most common type of skeletal tuberculosis and accounts for about 50% of bone and joint tuberculosis. It affects males and females equally. Though it is common in the first three decades of life, it can occur at any age.

It is popularly known as Pott's disease named after Sir Percival Pott, a successful British surgeon of the 18th century.

Development of Vertebral Column

By the 4th week in an embryo, paired cubical masses called somites start developing in the primitive mesenchyme. These somites differentiate into dermatome, myotome and sclerotome. Sclerotome surrounds the notochord. Each sclerotome starts segmentation and condenses into a cephalic mass, a caudal mass and a posterior element. The formation of the vertebra thus begins. Blood supply also follows this embryonic pattern. Thus, each segmental artery in the vertebral column supplies a caudal mass above and a cephalic mass below of adjacent provertebra respectively (In a fully developed vertebral column, the same pattern is maintained i.e. a segmental artery supplies lower half of one vertebra and upper half of the subsequent vertebra.) The notochord ultimately disappears in the region of the sclerotomes except in the region of the intervertebral disk, where it remains as nucleus pulposus. Nucleus pulposus along with annulus fibrosus, forms an intervertebral disk.

Clinical importance of this embryonic blood supply, is shown by the classical lesion in tuberculosis of the spine. It involves the lower half of upper vertebra and/or the upper half of the lower vertebra and manifests radiologically by narrowing of the disk space, (one of the earliest radiological signs). This also proves the fact that dissemination of tubercular focus occurs through the bloodstream.

Batson's Plexus of Veins

These veins connect pelvic veins to internal vertebral venous plexus. They do not have valves.

Hence, dissemination of infective focus from the genitourinary tract and abdomen to the vertebra, can occur easily.

Site of the Lesion

Lesion is common in lower thoracic spine followed by upper thoracic cervical, lumbar and sacral spines.

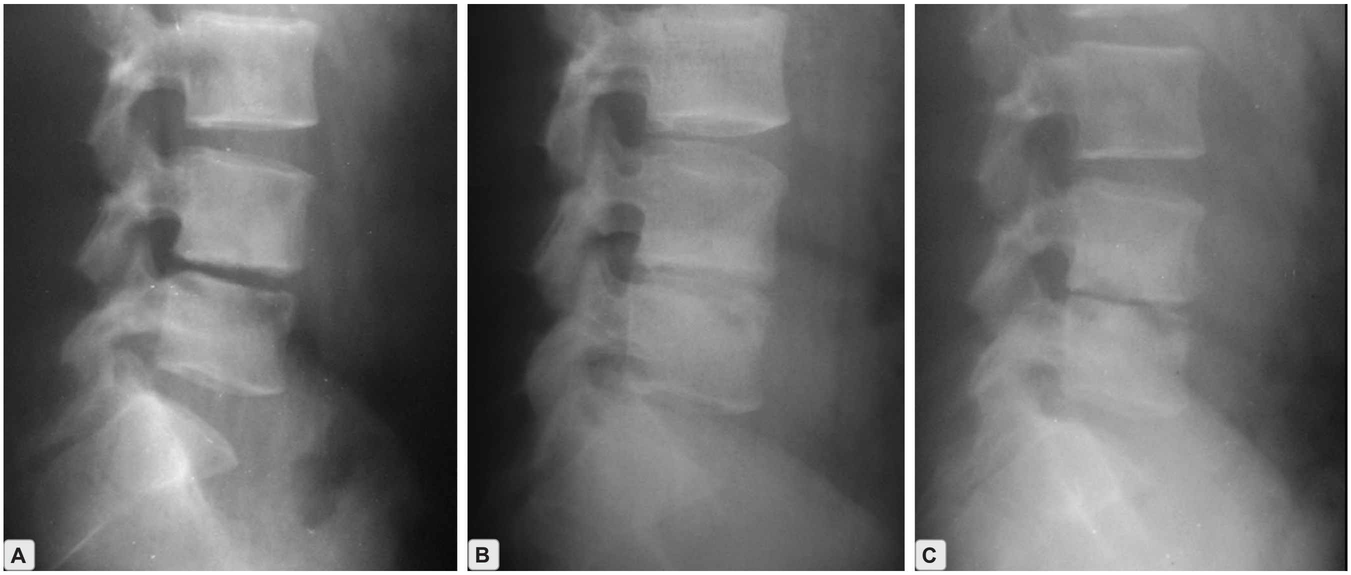
Probable reasons as to why lower thoracic spine is a common site:

- Batson's plexus of veins.
- Cisterna chyli begins at lower border of T12.
- Close relation of thoracic duct.
- Continuous movement with respiration which helps in dissemination.
- More spongy bone.

Vertebral Lesions

a. Paradiskal—Involves adjacent vertebra; intervertebral disk involvement is a late feature. Presents with narrowing of the disk space radiologically (>90%) (Figs 8.9A to C and 8.10).
b. Central*—Involvement of the body. Presents with cavity formation; no collapse of the body is seen radiologically, but a cystic lesion is seen.
c. Lateral*—Lateral segments of the body are involved. Shows lateral collapse of the body radiologically.
d. Anterior*— Anterior segments are involved. Shows anterior collapse of the body radiologically.

   *Vertebral body (central, lateral or anterior) involvement is around 5–7%.



Figures 8.9A to C

Radiographs taken at monthly intervals, at 1st, 2nd and 3rd months of presentation respectively. (A) Narrowing of the joint space which is the earliest classical sign of spinal tuberculosis; (B and C) Progressive narrowing of the joint space as well as development of bony lesions. Healing occurs in the form of interbody fusion.

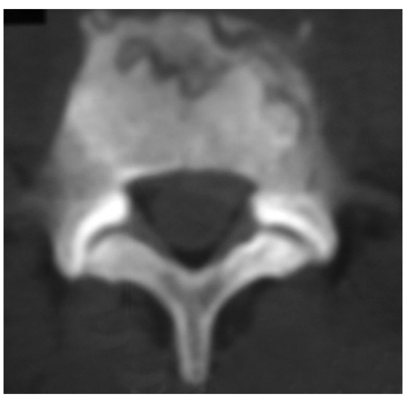


Figure 8.10

CT scan of 8.9A of the L5 vertebra taken in the 1st month at presentation. Note that the bony lesion can be identified much before the X-ray evidence.

e. Posterior elements—Articular facets, posterior spinal disease and spinal tumor syndrome (<5%).
f. Lesions involving multiple vertebrae and skipped lesions.

Clinical Features

a. Pain which is dull and vague initially becomes progressively severe and constant. It is aggravated by movement of the spine particularly jarring.
b. Loss of weight and appetite.
c. Fever and malaise with evening rise of temperature and night sweats.
d. Paraspinal spasm.
e. Presence of gibbus (angular, knuckle and round kyphosis).
f. Tenderness over the spine.
g. Presence of cold abscess.
h. Aldermanic gait—small steps with straight back to avoid jarring.
i. In cervical spine involvement, patient supports the chin over the palm of the hand.
j. Neurologic symptoms may be present.

Types of Kyphus/Gibbus

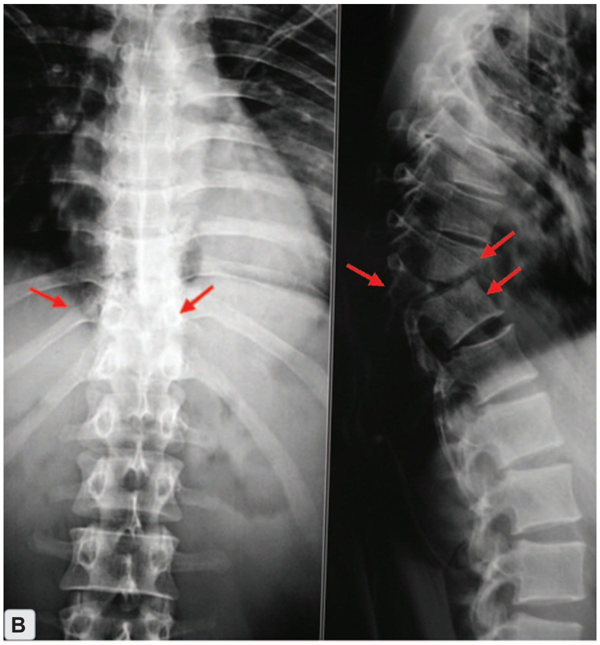
Involvement of adjacent portions of two vertebrae will result in 'Knuckle Kyphosis' (Figs 8.11A and B).

Involvement of three consecutive vertebrae will result in 'Angular Kyphosis' (Figs 8.12A and B).

Involvement of more than three vertebrae will result in 'Round Kyphosis'.

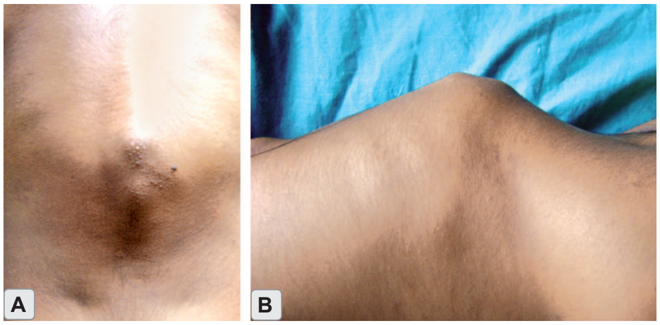
Cold Abscess

It is a collection of fluid and debris in tuberculosis. It is referred as 'cold' because it is devoid of signs of acute inflammation such as warmth, redness and pain. In the cervical spine, radiologically, the soft tissue shadow



Figures 8.11A and B

(A) Clinical picture and (B) Radiograph of an 'Knuckle kyphosis'. Note near total destruction of T11 and posterior subluxation. T10 is partially destroyed. Altogether two vertebrae are involved resulting in an 'Knuckle kyphosis' in the AP view note that the 11th rib is free of its attachment to the vertebral body because the vertebral body is destroyed almost completely.



Figures 8.12A and B

More than three vertebral involvement resulting in a round Kyphosis.

of a cold abscess appears like a bird's nest and in the mediastinum it is heart-shaped or fusiform.

Position and Course of Cold Abscess in Spinal Tuberculosis (Figs 8.13 to 8.15)

In any region

a. Within the prevertebral fascia as prevertebral abscess, in front of the vertebral body.
b. Within the spinal canal compressing the spinal cord.

In the cervical region

a. Retropharyngeal abscess (spreading cervical prevertebral abscess).
   Note: *Acute pyogenic abscess is in front of the prevertebral fascia and one side of the midline. Whereas, the cervical cold abscess is in the midline. This midline disposition changes when there occurs a break in prevertebral fascia.*
b. In the mediastinum-tracking down from the prevertebral region.
c. At the back of the neck-tracking along the posterior division of the spinal nerves.
d. At the posterior border of the sternomastoid muscle-tracking laterally.
e. In the axilla, through the open mouth of axillary sheath.

In the thoracic region

a. May remain as prevertebral abscess in the posterior mediastinum.
b. Through the openings in the diaphragm, namely, the lateral lumbocostal arch, the medial lumbocostal arch and median arcuate ligament (the two crurae of the diaphragm together from the median arcuate ligament) tracks distally, out of the thorax.

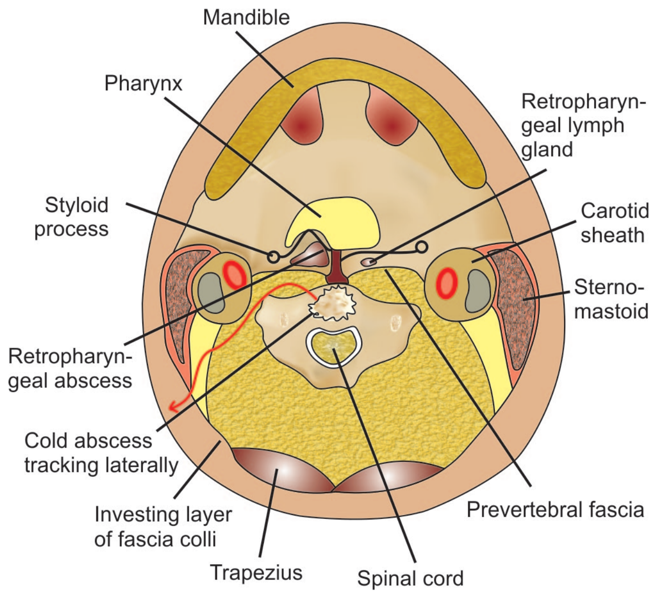


Figure 8.13

Path of spread of cold abscess in the cervical spine. Also shows the site of an acute retropharyngeal abscess.

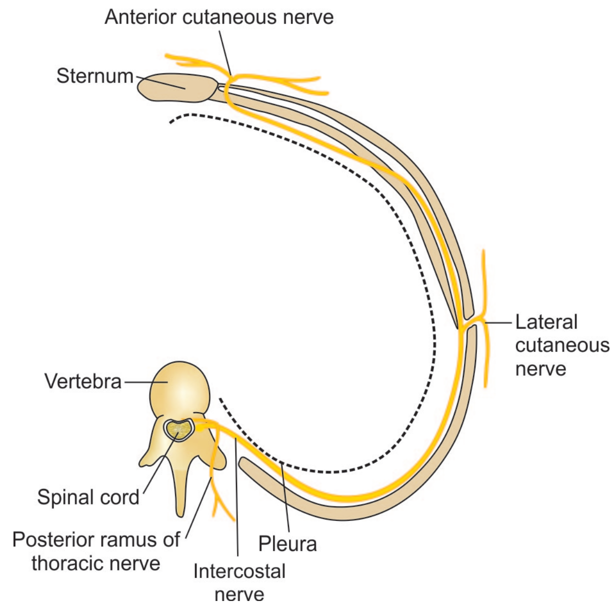


Figure 8.15

Lateral path for the spread of the thoracic cold abscess along the intercostal nerve.

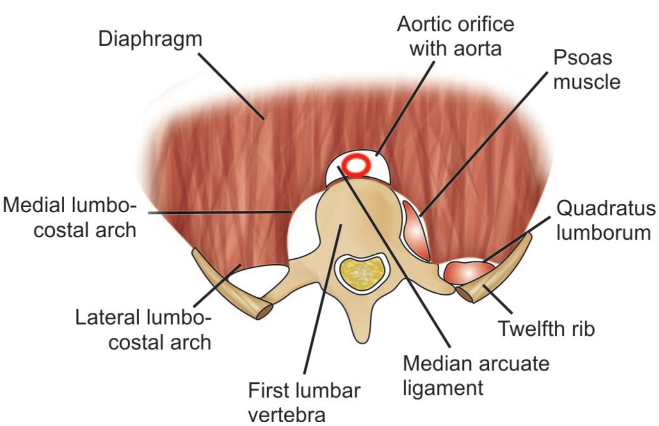


Figure 8.14

Paths of spread of the cold abscess in the thoracic region through the openings in the diaphragm.

- Lateral lumbocostal arch:
  - i. Traversing between the lumbodorsal fascia and quadratus lumborum muscle, may remain behind the kidney.
  - ii. Traversing along the 12th thoracic, ilio-hypogastric and ilioinguinal nerves, to the anterior abdominal wall.
- Medial lumbocostal arch:
  It is the open mouth of psoas sheath. It can track down to the insertion of psoas muscle into the thigh.
- Median arcuate ligament:
  The median arcuate ligament crosses over the aorta. So, the abscess may spread along the aorta and its branches.

c. Along the intercostal nerves, in the anterior middle or posterior axillary lines, in the chest wall.
d. In the rectus sheath, over the anterior abdominal wall.

In the lumbar region

a. Along the aorta and its branches, internal pudendal, superior gluteal, etc.
b. Along the sheath of psoas or quadratus lumborum.
c. Along the lumbar, femoral and obturator nerves into the thigh.
d. Into the 'lumbar triangle of Petit', through the flat muscles of the abdominal wall.

Investigations

- Hb%—low; TC—moderate increase; DC—predominantly lymphocytes; ESR—raised.
- X-ray—shows picture of destruction of the vertebra as per involvement. Narrowing of the intervertebral space, anterior collapse, lateral collapse, cavity formation, soft tissue shadow of cold abscess, etc. may be seen.
- CT, MRI—done for better evaluation of bone and soft tissue involvement.

Confirmatory tests

- Identifying presence of bacteria in the pus—Smears are made and stained for acid-fast bacilli.
- Classical tubercular granuloma is seen histopathologically under microscope in biopsy.
- Positive PCR (polymerase chain reaction) for tuberculosis in the pus sample.
- Pus culture and guinea pig inoculation—Takes a long time and not done regularly.

Pott's Paraplegia

When the thoracic spinal cord is involved in tuberculosis it results in paralysis of the lower limbs. In severe and late cases bladder and bowel also are involved. This is known as Pott's paraplegia named after Sir Percival Pott.

Sequential events of paralysis:

a. Muscle weakness
b. Awkward gait
c. Incoordination
d. Spasticity
e. Paraplegia in extension
f. Paraplegia in flexion

Types of Pott's Paraplegia

As proposed by Sorrel-Dejerine and later by Seddon.

A. *Early onset paraplegia:* Paralysis occurs during the florid phase of the disease, i.e. within 2 years.
B. *Late onset paraplegia:* Paralysis occurs many years after the disease, when the disease has become quiescent and sometimes even without reactivation.

Causes for Paraplegia as put forth by (Based on) Seddon

Early onset

i. Inflammatory
   a. Abscess, inflammatory tissue or a caseating mass.
   b. Spinal tumor syndrome.
   c. Posterior spinal disease.
   d. Infective thrombosis of the cord.
ii. Mechanical
   a. Pathological dislocation.
   b. Compression by sequestra, loose fragments of bone or disk, pressure of the granulation tissue and debris.

Late onset

i. Inflammatory: Persistent activity or reactivation of the disease.
ii. Mechanical: Progressive stretching of the cord ultimately jeopardizing its circulation.

In brief, the cause for paralysis is either the active disease process itself which damages the cord or the sequestrated material and structural abnormality which mechanically interferes with the function of the cord.

Treatment

Medical line of treatment: Always indicated when there is no neurological involvement. Drainage of the cold abscess is the only surgical procedure that may be necessary. It is chosen when investigations and initial assessment do not lead to absolute indications for surgery (indications for surgery as proposed by Griffths and Seddon are described in surgical line of treatment).

1. Antitubercular drug therapy (ATT) has to be started at earliest. It is author's firm belief that one should not hesitate to use injection streptomycin 0.75–1.0 gm daily, at least for 60–90 days whenever indicated. The site of injection has to be rotated daily (on four limbs) for maximum comfort. A minimum of three if needed four drugs have to be used to achieve an early effective control of the disease. The toxicity of the drugs should always be borne in mind during therapy and the therapy should be modified accordingly. Patient is to be evaluated on a daily basis during the initial period of therapy and if necessary liver enzyme study (SGOT/ALT, SGPT/AST, Gamma GT) and renal function tests may have to be done.

   Secondary infection if any should be taken care of by appropriate antibiotics.
2. Nutritional status is improved by giving good attention to the food intake.
3. The spine is immobilized in an appropriate brace or even in a plaster of Paris jacket when more rigid immobilization is indicated. This helps not only in giving comfort to the patient but also prevents development of severe deformities. Also helps in early consolidation of the diseased bone.
4. A watch is kept on signs of improvement (if no improvement is observed surgical treatment is indicated).

Surgical line of treatment: Single most important indication for surgery in tuberculosis is presence of paralysis.

Indications for surgery (based on indications as put forth by Griffiths and Seddon):

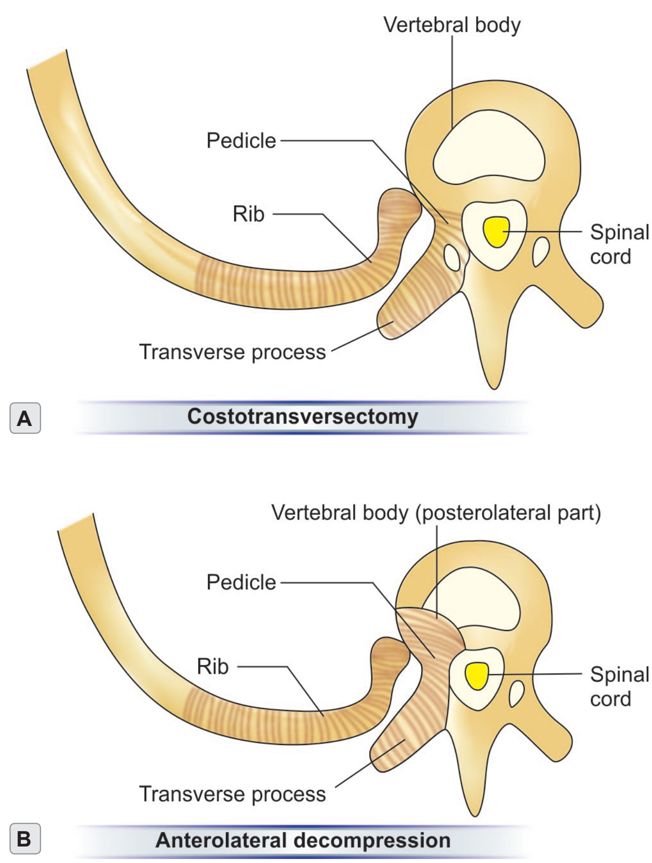
- *Absolute indications:*
  1. Paraplegia developing when the patient is on adequate medical line of treatment.
  2. Paraplegia not improving with adequate medical line of treatment.
  3. Paraplegia progressing even with adequate medical line of treatment.
  4. Complete paraplegia.
  5. Severe spastic paraplegia.
  6. Paraplegia of rapid onset.
  7. Long standing paraplegia > 6 months, paraplegia in flexion, flaccid paraplegia, etc.
- *Relative indications:*
  1. Recurrent paraplegia
  2. Paraplegia in old age
  3. Painful paraplegia
  4. Paraplegia with complications such as urinary infection and stones
- *Rare indications:*
  1. Posterior spinal disease
  2. Spinal tumor syndrome
  3. Cervical spinal tuberculosis
  4. Severe cauda equina paralysis

Surgical Procedures

1. *Drainage of abscess:* An abscess large enough for drainage is drained under cover of antitubercular drugs and antibiotics. Smaller abscesses resolve with adequate ATT.
2. *Costotransversectomy:* It is a surgical procedure for drainage of a large paravertebral abscess. The drainage is accomplished by excising the ribs, the transverse process and corresponding pedicle. A minimum of one and maximum of two ribs and transverse processes are excised (Fig. 18.16A).
3. *Anterolateral decompression:* It is a surgical procedure which decompresses the anterolateral portion of the spinal cord by excising posterolateral part of the vertebral body, corresponding ribs, transverse processes and pedicles. This is indicated when costotransversectomy does not yield pus under tension. The tubercular debris and granulation tissue is cleared by this procedure (Fig. 18.16B).
   Minimum of three and maximum of four ribs and corresponding transverse processes are excised.
4. *Anterior decompression and fusion:* It is a procedure employed when the lesion is anterior and anterior portion of the vertebral body is destroyed. Spinal fusion always follows decompression.
5. *Laminectomy:* Done only in cases of posterior spinal disease and spinal tumor syndrome.

Order of recovery of paralysis: The recovery of paralysis always takes place in the following order irrespective of modalities of treatment.

1. Vibration and joint sensation
2. Temperature, touch, pain
3. Voluntary motor activity
4. Sphincter functions
5. Wasting of muscles



Figures 8.16A and B

Portion of the bone removed (the shaded portion) in costotransversectomy and anterolateral decompression.

Factors governing prognosis:

1. Age: Younger the age, better is the prognosis and vice versa.
2. General condition: Good general condition, better is the prognosis and vice versa.
3. Duration: Shorter the duration, better is the prognosis and vice versa.
4. Severity: Incomplete paralysis without bladder and bowel involvement and sensory loss, better is the recovery and vice versa.
5. Type: Early onset, better is the prognosis and vice versa.
6. Speed of onset: Slow onset better is the prognosis than rapid onset.
7. Kyphotic deformity: More than 60° poorer is the prognosis.

Revision Questions

Q. Discuss the development of the vertebral column.

Q. Which is the most common site for spinal tuberculosis and why?

Q. What are the types of lesions found in the tuberculosis of the spine?

Q. What is a cold abscess? Discuss the natural mode of spread of cold abscess in different regions of the spine.

Q. Discuss the management of cold abscess.

Q. Write short notes on
  a. Bird's nest abscess.
  b. Vertebral lesions in tuberculosis.
  c. Battson's plexus of veins.
  d. Pott's paraplegia.
  e. Posterior spinal disease.
  f. Spinal tumor syndrome.

Q. Write notes on
  a. Anterolateral decompression.
  b. Costotransversectomy.
  c. Anterior decompression.

Essay Questions

Q. Discuss the pathogenesis, clinical features diagnosis and management of tuberculosis of the spine.

Q. Classify Pott's paraplegia. Discuss the management of Pott's paraplegia in detail and its prognosis.

Tuberculosis of the Hip

Tuberculosis of the hip is the next common site after tuberculosis of the spine. The disease is common during first 3 decades of life. The infection may get lodged as a solitary focus or as multiple foci during the phase of dissemination. They get lodged in the synovium or in the bones of the hip. In the bone, the site of involvement in the order of frequency is as follows (Fig. 8.17).

i. Roof of the acetabulum
ii. Superior subchondral portion of the femoral head
iii. Babcock's triangle
iv. Greater trochanter

It begins as tuberculous osteomyelitis. The foci of infection subsequently spreads to the joint and the articular cartilage gets destroyed. Thus, osteoarticular tuberculosis developes.

Babcock's triangle is a triangular area at the inferomedial portion of the neck of the femur, identified radiologically. Medially, it is bounded by the epiphyseal plate or by stress trabeculae; laterally, by primary compression trabeculae and inferiorly, by the neck of the femur (Figs 8.18A and B).

Clinical Features

- Low grade fever with evening rise of temperature and night sweats.
- Night pains.
- Loss of weight and appetite.
- Progressive painful limp and antalgic gait.
- Development of deformities.

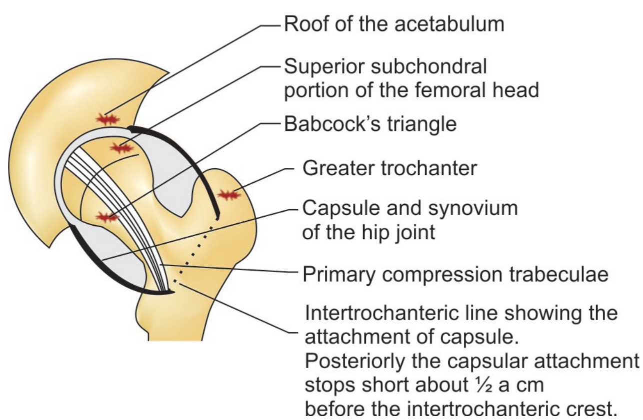
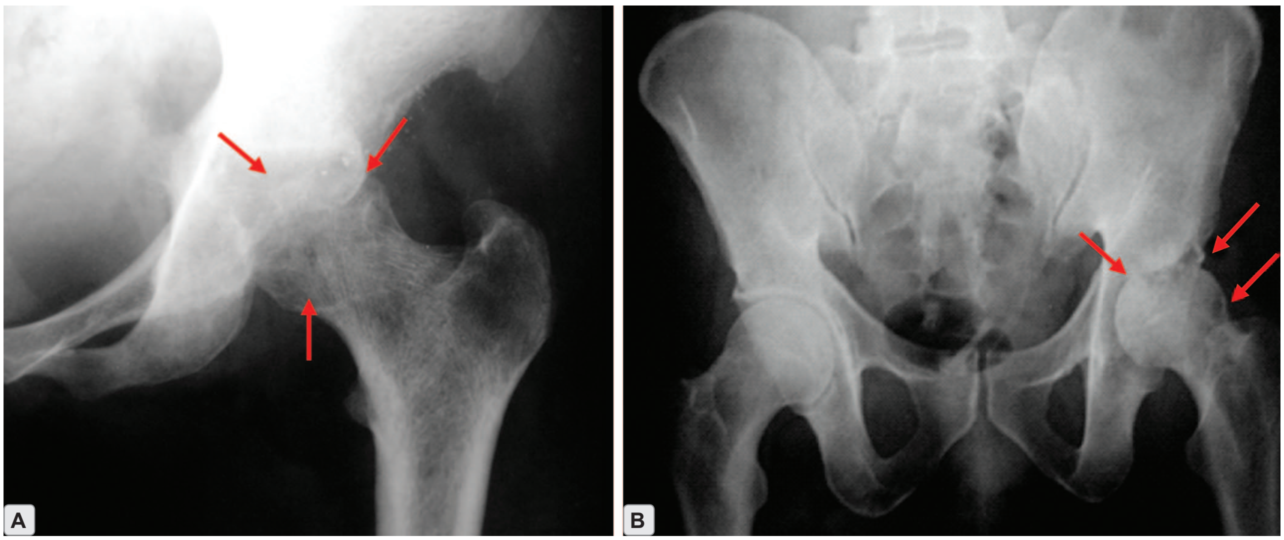


Figure 8.17
Common sites of foci of tuberculosis in the hip joint.

Stages of the Disease (Fig. 8.19)

Stage I—Stage of synovitis (Apparent lengthening) In this stage, the focus of infection (whether in the synovium or in the bone) irritates the joint. Hence, there is collection of fluid in the joint. In order to accommodate the fluid the joint adopts a position which increases its capacity. Hence, the limb is in flexion, abduction and external rotation.

Stage II—Stage of arthritis (Apparent shortening) In this stage, the osteoarticular spread of infection has occurred. The joint destruction has begun and is progressing. Thus, intensity of pain is severe and patient sleeps in a lateral position, opposite to that of the involved hip. Hence, the deformity of flexion, adduction and internal rotation develops. Night pain is common in this stage.



Figures 8.18A and B

(A) Narrowing of the joint space with a lesion in the 'Babcock's triangle. Patient was a known case of pulmonary tuberculosis and was not taking regular treatment. Also, note the soft tissue swelling around the joint. Skeletal tuberculosis is almost always secondary to a primary lesion elsewhere in the body; (B) Classical tubercular lesions in the left hip, in a middle aged man, in the roof of the acetabulum and superior subchondral portion of the head of the femur.

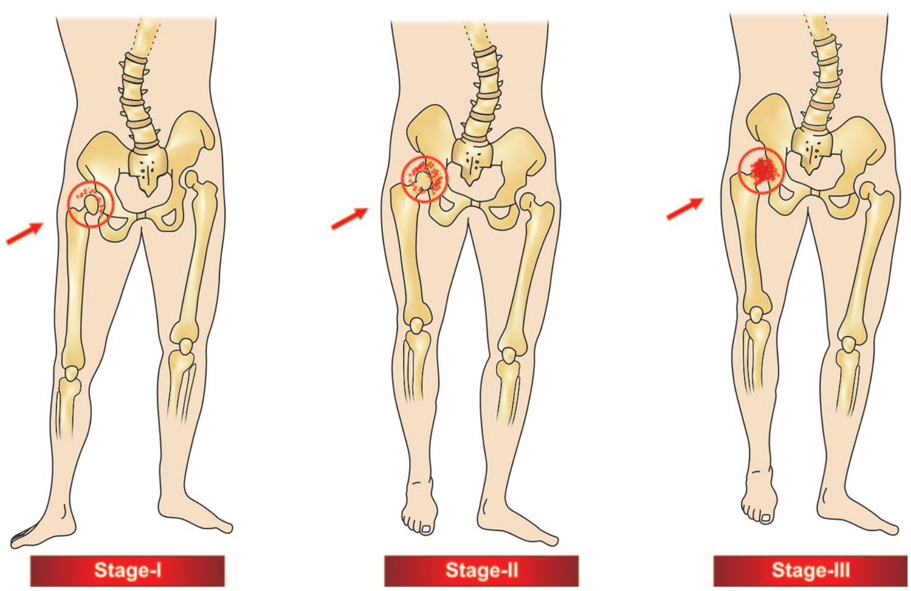


Figure 8.19

Deformities in three stages of tuberculosis of the hip joint. Stage I: There is apparent lengthening because of abduction deformity; Stage II: There is apparent shortening because of adduction deformity; Stage III: True shortening develops because of destruction of the joint.

Night pain/Night cries: Patient gets up from sleep at night with severe and excruciating pain. Once he is awake, the pain gradually decreases. This is known as night pain. It occurs because, the denuded articular sufaces (denuded of its articular cartilage) come into contact with each other and the exposed subchondral bone rubs against its counterpart. During daytime, protective muscle spasm prevents this contact. But at night, in sleep when the muscles are relaxed, the contact develops. Again, when the patient gets up from sleep, the protective muscle spasm keeps the denuded articular surfaces away and the pain decreases. This phenomenon is known as night pain.

Stage III—Stage of deformity (True shortening) In this, the deformity of second stage becomes fixed. Destruction is pronounced and the joint goes into a state of fibrous ankylosis. The classical deformity is that of fixed flexion, adduction and internal rotation. But, if the Y ligament of Bigelow is destroyed in the process of infection, it is possible to get a deformity of flexion, abduction and external rotation.

Radiological Classification

An excellent classification is suggested by Professor Shanmugasundaram, Chennai (1983), India (Figs 8.20A to C).

Normal hip type Perthes' hip type Dislocated hip type	} Seen in children
Atrophic hip type	Seen in adults
Traveling acetabulum type Mortar and pestle type Protrusio acetabuli type	} Seen both in children and adults

Functional outcome is different in different types.

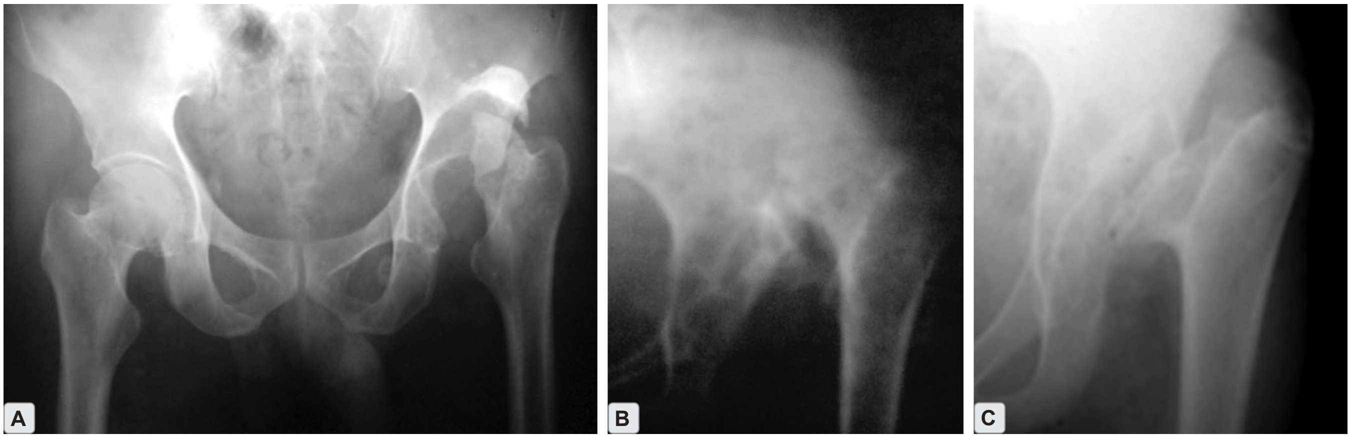
Treatment

General: Adequate ATT till the disease is cured is absolutely necessary. Immobilization in a splint or a plaster cast only for the required period is a must. This will help to restore the functional position of the joint and prevent unacceptable deformities. As soon as possible, depending on the recovery, the patient has to be mobilized. This will avoid all the complications of unnecessary prolonged immobilization.

Surgical Procedures

Clearance procedure: This procedure is indicated early in the disease when the response to medical line of treatment is poor because of the presence of large amount of debris. It aims at salvaging the joint by extensive debridement of the infected material and dead tissue thereby facilitating the action of drugs of ATT. At the same time, the joint is immobilized in desirable functional position in order to facilitate healing in a position of maximal function.

Arthrodesis: It is indicated in a case of unsound ankylosis of the hip, where stability is of prime concern. Functional position of arthrodesis is about 5–10° of flexion, about 10° of abduction and 0–5° of external rotation. Disadvantage is the inability to squat in future.



Figures 8.20A to C

Different types of presentation based on Professor Shanmugasundaram's description of tuberculosis of the hip joint: (A) Mortar and pestle type; (B) Traveling acetabulum type; (C) Atrophic type.

Hence, all those activities which involve squatting have to be modified. The procedure gives a painless stable hip. Procedure should never be done in children.

Osteotomy: This procedure is done in a case of bony ankylosis which has occurred in a functionally poor position. The osteotomy is done as close to the deformity as possible in the femur (just distal to the deformity) and the deformity is corrected.

Excision arthroplasty: The procedure involves excising all the diseased components including the head of the femur in an attempt to provide a painless mobile hip. Postoperatively, skeletal traction is employed for a period of 6–12 weeks for organization of a false joint. Exaggerated painless movement, instability and gross shortening are the disadvantages. This procedure is known as Girdlestone procedure. It was being done in the past in patients who preferred a painless mobile hip and did not mind shortening. This procedure is rarely done these days.

Joint replacement surgery: Total joint replacement is the preferred option these days. At times, the acetabulum may have to be reconstructed. The procedure is well accepted by the patient and it gives a painless mobile hip. Patient should be totally free of infection which is the prerequisite before surgery.

Prognosis

Early presentation, diagnosis and adequate control of the disease give better prognosis than late presentation and delay in the treatment, i.e. stage of synovitis has better prognosis than stage of arthritis and fixed deformities. Once the destruction occurs in the joint, the end-result is fibrous ankylosis.

Tuberculosis of the Knee

Tuberculosis of the knee is the third common site after spine and hip.

Similar to any other bone and joint tuberculosis, the dissemination occurs through the bloodstream. The foci of infection get enlodged in the synovium or in the subchondral bone of the distal femur, proximal tibia or patella.

Unlike other joints, since the knee joint has the largest joint space and has large amount of synovial covering, the disease remains as synovial for a considerable time before it becomes osteoarticular. Thickened synovium, studded with nodules is visible to the naked eye during surgery. This is the result of invasion by tuberculous granulation tissue. The synovial fluid which is serous initially, becomes seropurulent and turbid later and may contain fibrinous flakes.

The synovial disease becomes osteoarticular by the formation of pannus. The pannus starts eroding the bone at the junction where the synovial membrane is attached to the articular cartilage, the capsule and the ligaments. The pannus can extend over the articular cartilage as well as under it thereby separating it from the subchondral bone. This results in flakes of cartilaginous debris being present within the joint.

When the disease starts as osteoarticular, the pathology is that of tubercular osteomyelitis.

Generally, the location of the lesion is epiphyseal in adults and metphyseal in children. The cold abscess thus formed destroys the bone and the articular cartilage, and finally, enters the joint.

Pathology

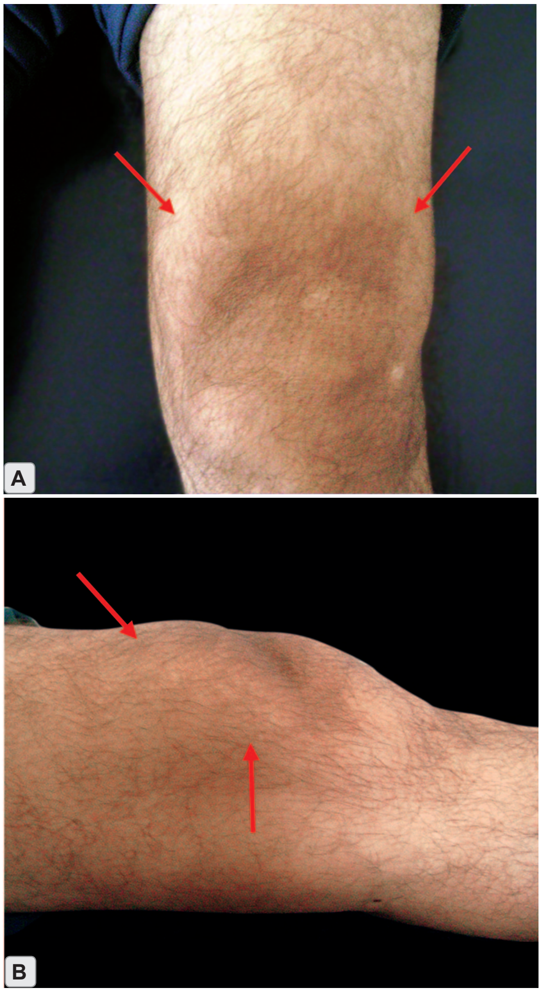
Early lesions are seen in synovium and bone (Figs 8.21 and 8.22). Later, disease destroys the joint and progresses to tuberculous arthritis (Fig. 8.23). Fibrous ankylosis is the terminal event (Fig. 8.24).

Triple deformity: Whether synovial or osteoarticular, total destruction of the joint along with its capsule and ligaments (the cruciates and the collaterals, which is a late feature) leads to classical triple deformity, i.e. flexion + posterior subluxation + lateral subluxation, rotation and abduction.

Initially, flexion deformity develops because the knee adopts a position of flexion to accommodate the debris and the fluid. If it remains in flexion for a long time, posterior subluxation develops. The powerful pull of the iliotibial band and the hamstrings causes rest of the displacements, i.e. lateral subluxation, rotation and abduction.

Clinical Signs and Symptoms

- Low grade fever with evening rise of temperature and night sweats.
- Loss of weight and appetite.
- Progressing painful limp and antalgic gait.
- Effusion and synovial thickening.
- Wasting of the muscles around the knee mainly the quadriceps.
- Night pains.
- Development of deformities (late feature).



Figures 8.21A and B

Synovitis with effusion. Note the horse-shoe shaped swelling in the suprapatellar pouch obliterating the normal hollows around the knee joint. This is one of the earliest signs seen in TB knee (arrows).

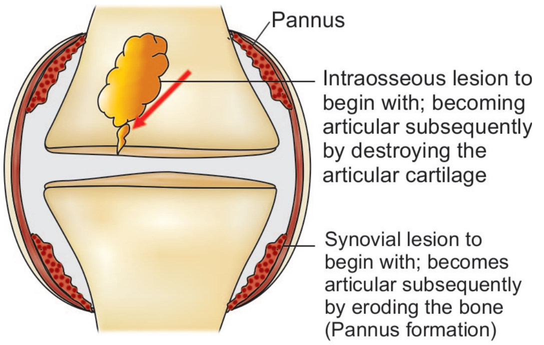


Figure 8.22

An osseous/synovial lesion subsequently destroys the joint and causes tubercular arthritis. The end result is fibrous ankylosis. Only if secondary infection sets in, the joint goes in for bony ankylosis.

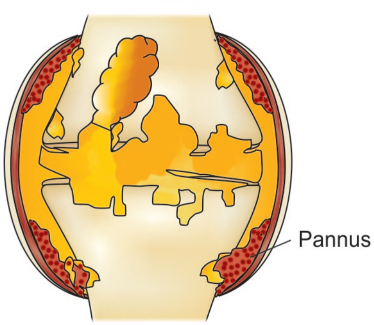


Figure 8.23

Destruction of articular cartilage and the involved bone. This results in loss of joint congruity.

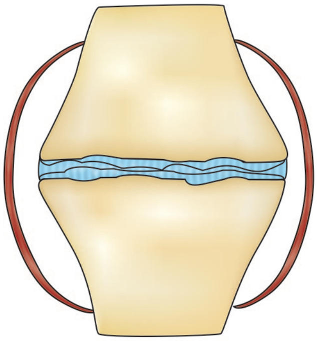


Figure 8.24

End result. The joint space is narrowed. The area of denuded articular surfaces are opposed with a bridge of fibrous tissue (fibrous Ankylosis).

Investigations

Blood and other investigations are similar as in any other bone and joint tuberculosis and give positive indications of the disease.

X-ray—depending on the stage of the disease it shows several features varying from soft tissue prominence to narrowing of joint space, destruction of the articular surface, osteolytic cavities, sequestrum and triple deformity (Figs 8.25A and B).

Computed tomography (CT) and MRI give more useful information for planning treatment and surgery.

Treatment

Medical management is indicated only in very early cases where synovial involvement is not extensive, which are diagnosed and confirmed by biopsy (arthroscopy and biopsy/trochar biopsy). ATT and adequate immobilization results in cure. In children, whose growth

Figures 8.25A and B

(A) Clinical picture and (B) Radiograph of the triple displacement showing classical displacements of flexion, lateral rotation and posterior subluxation.

and remodeling capacity is very high, medical line of management is to be always kept in mind before planning surgery.

Surgical Procedures

Synovectomy: Done in cases with grossly thickened synovium. It aims at removing the diseased synovium and clearing the joint of tubercular debris. Though total synovectomy is aimed at, practically it always results in subtotal synovectomy. This is because, in some areas, the synovium is inaccessible for surgical excision.

Arthrodesis: Done for a destroyed, deranged and deformed joint which is painful. The position of arthrodesis is 180° of extension. Charnley's compression arthrodesis using Charnley's compression clamps is one of the best methods for arthrodesis.

Total joint arthroplasty: Is the best accepted procedure. To be considered in adults and elderly. It gives a painless mobile joint with considerable function, when muscle atrophy is not very severe. Patient's disease free status should be confirmed before surgery to avoid flare-up of the disease.

Tuberculosis of the Ankle and Foot

Not very common. Focus of infection may be synovial or bony, i.e. lower tibia, talus and calcaneum are the commonly involved bones. Infection can spread to the ankle joint from calcaneal tuberculosis after involving the subtalar joint. Diagnosis and treatment follow the general principles as discussed before.

Tuberculosis of the Shoulder and Elbow

Shoulder: Synovial type of tuberculosis of the shoulder is very rare. So is the formation of cold abscess and draining sinus. When it occurs, the nature of tuberculosis of the shoulder is of a dry variety and is known as 'Caries Sicca'. It is commonly seen in adults and is considered as an atrophic variety of tuberculosis. Pain, limitation of movement and marked wasting of the muscles draws attention. Radiologically, destruction of the articular surfaces and cavities are seen. Management follows the general principles as discussed earlier in the chapter.

Elbow: The focus of infection in tuberculosis of the elbow is mostly bony and rarely synovial.

The disease is commonly seen in older children and adults. The signs and symptoms are similar to any other bone and joint tuberculosis. Diagnosis and treatment follow the general principles.

Tuberculosis of the Wrist

Tuberculosis of the wrist is rare. The incidence is more in adults when compared to children. May start as a synovial or a bony focus. Common sites are capitate

and lower end of radius. Both synovial and bony foci, disseminate to neighboring structures rapidly. The flexor and extensor tendon sheaths also get involved. The signs and symptoms are similar to any other bone and joint tuberculosis. Diagnosis and treatment follow the general principles.

Spina Ventosa (Tuberculous Dactylitis)

Spina, means short bones, *Ventosa* means air. Thus, the term means 'short bone filled with air'. Also known as tuberculous dactylitis. Commonly seen in children, in short tubular bones such as the metacarpals, metatarsals and phalanges. The blood supply of these bones is very rich and point of entry of the nutrient artery is in the center. Hence, the focus of infection gets lodged in the center. Central destruction occurs initially. This gives the picture of 'bone filled with air'. Later, layers of subperiosteal new bone form. This causes a spindle shaped expansile lesion. Sinus formation can lead to secondary infection and the picture changes to that of non-specific osteomyelitis. Adequate ATT with antibiotics in case of secondary infection leads to complete resolution.

Revision Questions

Q. Discuss the onset and progression of lesions in tuberculosis of the hip.
Q. Write notes on Babcock's triangle.
Q. Discuss the types of deformities in tuberculosis of the hip joint.
Q. Discuss the different radiological types of tuberculosis of the hip joint.
Q. Discuss the management of the tuberculosis of the hip joint.
Q. What is the sequel of the tuberculosis of the hip joint?
Q. Discuss the pathology of tuberculosis of the knee joint.
Q. Write notes on:
   a. Spina ventosa
   b. Triple displacement
   c. Fibrous ankylosis
   d. Clinical features of tuberculosis of the knee joint.

Essay Questions

Q. Discuss the etiopathogenesis, clinical features, diagnosis, and management of tuberculosis of the hip including the sequel.
Q. What is triple displacement? Discuss the etiopathogenesis, diagnosis and mangement of tuberculosis of the knee.

Mycotic Infections of the Bone

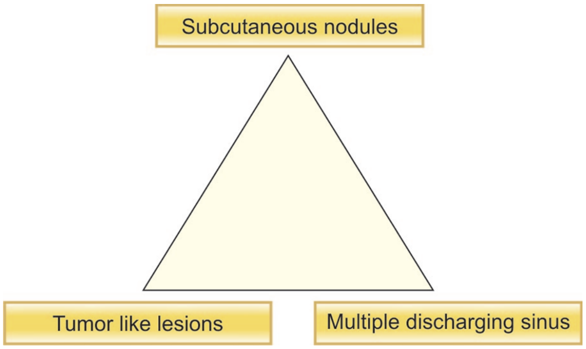
The Mycotic infections that involve the bone can be of two types.

i. Subcutaneous
   These infest subcutaneous tissue then spread to the bone
   e.g. *Madurella mycetomatis,* etc.
ii. Systemic
   These spread through the blood stream.
   e.g. *Aspergillus, Candida*,

Maduromycosis

First identified in Madurai, India in the year 1842. Thus named as Madura foot.

Caused by *Madurella mycetomi*, and *Madurella grisea*. Agricultural workers and those who walk on bare foot are more susceptible. Causes nodular lesions, once infection gets established in the tissues. Hence, derives the name mycetoma.



Route of entry is through skin following a cut wound. Spreads through subcutaneous tissue, the fascia and the tendon sheath and later gets established in the bone and joint. It evokes a granulomatous reaction in the tissues involved which break up causing multiple ulcerations and formation of multiple sinuses. The surrounding area is indurated and gives a woody feel.

Diagnosis is established by identification of the organism in the biopsy specimen and the sinus discharge.

Drug therapy with trimethoprim-sulfamethoxazole, ketoconazole, dapsone, etc. are effective only in early cases. Severe forms of involvement necessitate amputation.

Syphilitic Infection of the Bone

Seen in infants as well as in adults as a tertiary lesion. In infants it manifests after several weeks of birth though infection has occurred during pregnancy in the womb. *Treponema pallidum* crosses the placental barrier in the later months of pregnancy and infects the fetus. An irritable infant with painful, symmetrical skeletal swelling in the long bones and refusal to move the limbs should raise the suspicion. The pathology is either syphilitic periostitis or metaphysitis. Periostitis manifests radiologically by formation of new bone either diffuse or in layers. Metaphysitis shows trabecular erosion and frank destruction.

In older children and adults, gummatous lesions develop in the bone. Radiologically, seen as lytic punched out areas in the medulla surrounded by thick sclerotic bone. In the tibia, syphilitic osteitis causes diffuse thickening of the cortex and bending/bowing of the tibia which is classical. The resultant deformity is known as 'Sabre tibia'.

Parasitic Infestation of the Bone

Hydatid Cysts in the Bone

The dog tapeworm *Echinococcus granulosus* (5 mm long with 3 segments) is responsible for hydatid cysts in the bone. Dog is the definitive host and the tapeworm harbors in the bowel. The scolices are excreted in the feces. Sheep and cattle are intermediate hosts and get infested by ingesting food contaminated with dog feces. Infested meat when consumed by a dog continues the lifecycle and a new generation of tapeworms evolve.

Man is an accidental host. The scolices when ingested, hatch into larvae and through the portal system enter the bloodstream. And through the bloodstream may get enlodged in the bone leading to hydatid cysts. The common bones affected are the pelvis, vertebrae, ribs and femur. The cyst goes on destroying the bone. The growth plate does not form a barrier for destruction and the destruction may extend even to the epiphysis.

Radiologically, solitary or multiloculated cystic lesions are seen with extensive destruction of cortex and medulla. In case of vertebral involvement, patient may present with neurological signs. CT and MRI help in diagnosis by identifying para-skeletal cysts. Needle biopsy and Casoni's complement fixation test were confirmatory tests of the past. These days, serological tests such as indirect hemagglutination is done for confirmation.

Albendazole is the drug of choice and is to be given at the dose of 400 mg BD with meals for 4 weeks. Repeated after 14 days (2 weeks). A total of 3 cycles may be necessary. Protection may be necessary to avoid pathological fracture. Once the control is established, the bone cysts may need excision and bone grafting. Irrigating the cavity with hypertonic saline helps in preventing recurrence.

Revision Questions

Write notes on

a. Mycotic infections of the bone
b. Hydatid bone disease
c. Sabre tibia

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9 Bone Tumors

- *Evaluation of bone tumors in general with relevant investigations*
- *Classification*
- *Common benign bone tumors, their clinical features, diagnosis and treatment*
- *Common malignant bone tumors, their clinical features, diagnosis and treatment*

Bone tumors benign or malignant, can arise from any tissue (cells) present in the bone. Namely, bone and cartilage progenitor cells (most common), periosteal cells, hemopoietic cells, lipocytes, nerve and schwann cells, fibroblasts, endothelial cells, perithelial cells, epithelial cells, smooth muscle cells, notochordal cells, histiocytic cells, etc. They exhibit a specific predilection to age, specific bones and in the bones to specific sites. The nature of lesion is either to form neoplastic new bone or to destroy the normal bone, i.e. either tumor osteogenesis or tumor osteolysis (Figs 9.1A to C). Tumor osteolysis is generally seen when the bone tumor arises from elements other than bone and cartilage progenitor cells. Predilection to sex is also seen and generally males are more commonly affected when compared to females. All these points are to be kept in mind during evaluation of a bone tumor.

EVALUATION OF A BONE TUMOR

Clinical Evaluation

Apart from age, sex, location and the bone from which the tumor is arising, following are to be considered for primary evaluation.

Onset

Benign tumors: Onset is insidious in benign tumors. The first sign is development of a well-demarcated swelling with or without accompanying tenderness, i.e. in neoplastic new bone forming variety. It can also be an accidental discovery with no symptoms. At times, presentation is a pathological fracture with pain, i.e. in osteolytic variety. Further, constitutional symptoms are uncommon in benign tumors unless secondary complications such as malignant transformation, adventitious bursitis, etc. develop.

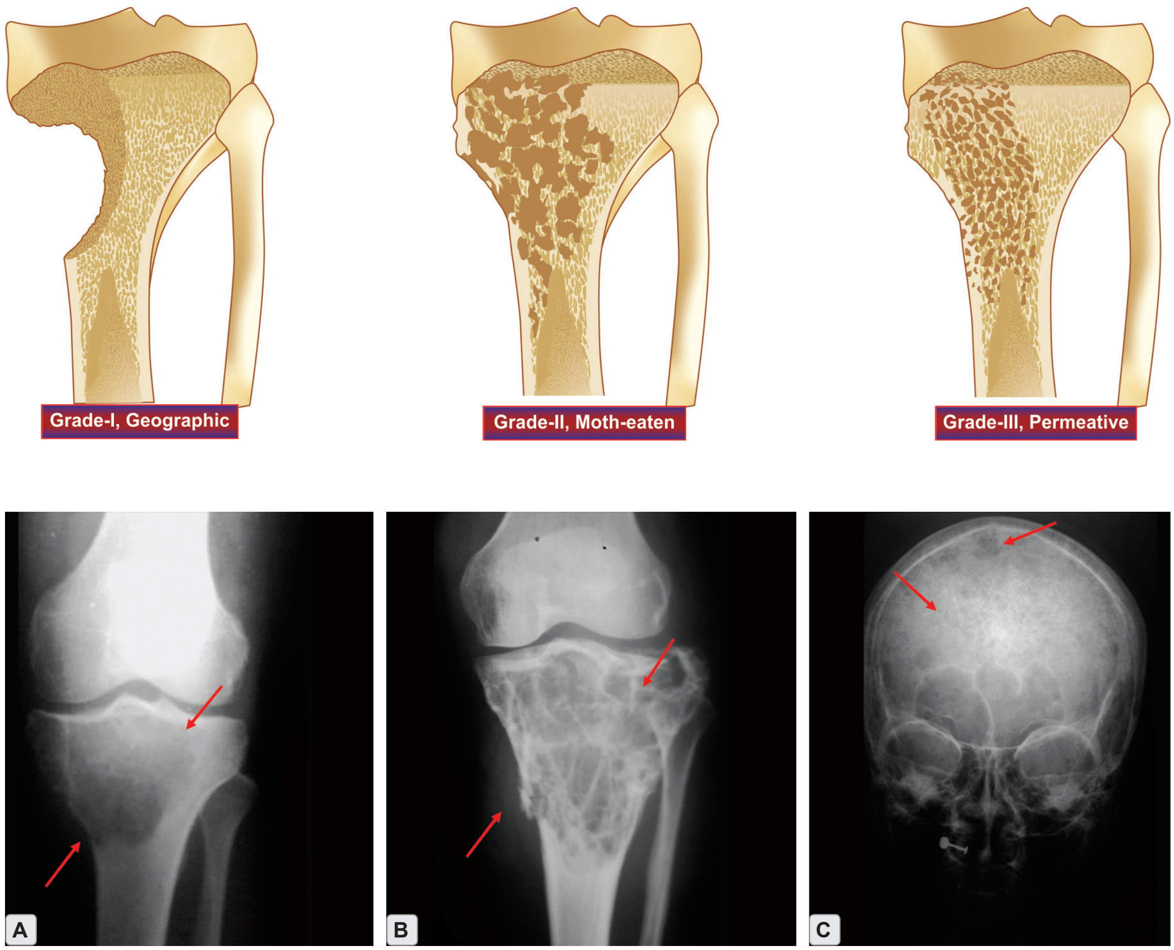
Malignant tumors: In malignant tumors, pain always precedes swelling. Most of the time, these tumors are associated with constitutional symptoms such as malaise, low grade fever, loss of appetite, weight, etc. Tumor related pain is constant and not relieved by rest.

Plane, Nature and Consistency of the Swelling

Plane of the swelling is always deep to the muscle as bone tumors arise from the bone. In benign lesions, the swelling is regular with a smooth surface and is of uniform consistency. Whereas in malignant lesion the swelling is irregular with variegated consistency (a malignant tumor may show different consistencies in different areas). Consistency varies between soft, firm and hard depending on the predominance of the tissues involved, as well as the amount of tissue destruction/ formation occurred due to the tumor.

Rate of Growth

Rapid in malignant bone tumor. Slow in benign bone tumor.



Figures 9.1A to C

Diagrammatic representation and radiographs of the types of destructive lesions (lysis) based on description by Lodwick. (A) Grade-I—Geographic (sharply defined border); (B) Grade-II—Moth-eaten (ragged border); (C) Grade-III—Permeative (wide zone of transition).

A general classification of bone tumors (depending on tissue of origin)

Nature	*Benign*	*Malignant*
A Angiod tumors	Angioma, ABC Glomus tumor	Angiosarcoma
B Bone forming tumors	Osteoma Osteoid osteoma Osteoblastoma	Osteosarcoma
C Cartilage forming tumor	Chondroma Osteochondroma Chondroblastoma	Chondrosarcoma
D Dental and allied structures	Odontogenic cyst Amelloblastoma	Malignant odontoma

Nature	*Benign*	*Malignant*
E Embryogenic vestigeal tissue		Chordoma
F Fibroblastic tissue	Fibroma	Fibrosarcoma
H Heterotropic tissue	Dermoid	Adamantinoma of long bones
N Non osseous connective tissue	Lipoma Neurofibroma Neurilemmoma	Liposarcoma Myeloma Leukemia, Ewings
S Synovial tissue	Synovioma	Synovial sarcoma
U Undifferentiated	Giant cell tumor	Malignant Osteoclastoma

Extension into Surrounding Soft Tissue

Is a feature of malignant bone tumor e.g. 'Satellite' and 'Skipped' lesions. It is not a feature of benign bone tumor.

Regional Lymph Node

Lymph nodes are enlarged with firm/hard consistency in malignant tumors suggesting lymphatic spread. Lymph nodes are not enlarged in benign tumors.

Radiological Evaluation

A radiograph is viewed as follows.

a. Age of the patient: Certain tumors are common in certain age groups, e.g. Ewing's tumor 5–15 years, Osteosarcoma 15–25 years, Giant cell tumor 20–30 years, etc. Hence attempt should be made to ascertain the age of the patient radiologically.
b. Bone involved: Certain tumors are common in long bones and in appendicular skeleton, while others are common in flat bones and axial skeleton, e.g. osteosarcoma is commonly seen in long bones, multiple myeloma is commonly seen in flat bones and the vertebra.
c. Site in the bone

   Long Bone
   - Epiphysis: Giant cell tumor.
   - Metaphysis: Osteosarcoma.
   - Diaphysis: Ewing's tumor.

   Vertebra
   - Body: Primary tumor.
   - Appendages: Secondary deposits.

d. Nature of the lesion

   Osteogenic: Osteoid osteoma, Osteosarcoma

   Osteolytic: Osteoblastoma, Multiple myeloma
e. Specific characteristics

   Soap bubble appearance: Giant cell tumor.

   Onion skin appearance: Ewing's sarcoma.

   Speckled calcification: Chondrosarcoma.

   Punched out appearance: Multiple myeloma.

Bone scan: Bone scan using Technetium 99 m diphosphonate (99 m Tc-HDP) is of immense help as it clearly shows the hot spots in the skeleton. Even the smallest of the tumors can be diagnosed and so also the skipped metastasis.

CT scan: CT scan is not a substitute for radiographs or bone scan, but it is an adjuvant to these investigations, when there is a need for clear delineation of tissues. It is useful to assess the intra and extraosseous extension of the tumor. It also helps in early detection of tumors before radiological changes are seen.

MRI: MRI delineates the soft tissues better than CT and is useful in visualizing the soft tissue extension and invasion of the tumor into the neighboring structures, e.g. muscles, vessels and nerves. This helps in staging the tumor and planning the surgery.

PET scan (positron emission tomography): It reveals how the body part is functioning unlike CT and MRI which simply give an image. It is not only useful in early diagnosis but also useful in evaluating response to treatment.

A radioactive tracer FDG—Fluoro deoxyglucose is inserted into the human body. This molecule of glucose tagged with the radioactive tracer, is utilized by the tissues for energy and as it breaks down it emits positrons. The gamma rays emitted indirectly by the positrons, are detected by the machine and a 3D color image is reconstructed. The image reveals the functional process going on in the human body by detecting the metaboilc changes occurring at the cellular level. The diseased cell utilizes glucose in a different manner than the normal cell. Hence, the image obtained is a functional image which helps in early diagnosis of a disease. It is also useful in evaluating the response to treatment. All the modern PET machines allow a CT image along with PET scan, simultaneously. So, the investigation is known as PET CT.

The disadvantage of PET is that it is almost 5 times more expensive than MRI and almost 8 times more expensive than a Technitium 99 M bone scan.

Note: *A by product of FDG, i.e. F18 is being effectively used for bone scan. This reduces the cost of bone scan.*

Role of Angiography

Angiography has a role to play when there is a need to asses the vascularity of the tumor and to localize the vessel when a radical surgical procedure is being planed. Sometimes, the vessel may get involved in the tumor tissue and separation becomes difficult during surgery. Hence, a need arises to sacrifice some of the branches. In such cases, preoperative angiography is useful in determining the presence of good and adequate vascular anastomosis which can salvage the limb, e.g. brachial artery involvement at the elbow, popliteal artery involvement around the knee.

Angiography also helps in identifying skipped lesions when used with other imaging techniques, e.g. CT angio.

After the clinical examination, investigations and confirmation of the diagnosis, a conclusion has to be drawn with respect to the exact status of the tumor. This dictates the treatment. Benign tumors do not pose much problem. A total excision results in a cure. But a malignant tumor poses a challenge as it may recur or cause metastases. Enneking system of staging (1980) helps in taking decisions with respect to the treatment of a malignant tumor.

Enneking Staging

I—Low-grade tumor

A—Intracompartmental

B—Extracompartmental

II—High grade tumor

A—Intracompartmental

B—Extracompartmental

III—Either grade with distant metastasis

Accordingly, a curative or palliative treatment is planned. A biopsy of the lesion is a must before surgery.

Bone Biopsy

Bone biopsy is done to confirm the clinical diagnosis with certainty. It gives an accurate diagnosis as well as the aggressiveness of the tumor, etc. When sections studied show increased atypical features, the tumor is considered more malignant. Open biopsy procedues of the past have been replaced by more refined procedures, e.g. needle and core biopsy. These less invasive procedures, avoid spillage of the cells and thereby prevent seeding of the surrounding tissue with malignant cells. Thus, they facilitate limb salvage in malignant bone tumors.

Indications for Biopsy

1. Benign aggressive leisions
2. Malignant lesions
3. Lesions with uncertain clinical diagnosis

Contraindication

1. Benign lesions (Excision biopsy indicated)

Note: *Most of the lesions these days are diagnosed by a good clinical history, physical examination, radiologic imaging studies and laboratory data. Biopsy is only confirmatory.*

The following information helps in appropriate planning and execution of a biopsy.

Basic Information

Most of the malignant bone tumors are of mesenchymal origin and are sarcomas. They grow in a centripetal fashion. Most immature part is found at the growing edge of the tumor. A reactive zone is formed between the tumor and the compressed normal tissue surrounding the tumor. This zone is mainly composed of neovasculature and inflammatory cells.

Satellite lesions: When microextension of the tumor is found in the reactive zone, the lesions are known as 'Satellite lesions'.

Skipped lesions: When lesions are found in the same anatomic compartment but outside the reactive zone they are known as 'Skipped lesions'. They are common in high grade sarcomas and rare in low grade sarcomas.

Note: *Sarcomas push the surrounding tissues and form a reactive zone whereas carcinomas do not. They infiltrate the surrounding tissues.*

Specific Information

1. *Sampling error:* This occurs when the biopsy is taken from a region other than the primary disease, e.g. reactive zone. To avoid this, the anatomic location of the biopsy site should always be planned well before the procedure. Suspected necrotic area is to be avoided for better yield.
2. *Incision:* When open biopsy is planned the incision should always be in an area of resection of a future definitive surgical procedure. If a drain is kept, the drian track should also be placed in a similar manner.
3. *Staging:* Biopsy is always planned after the imaging studies are complete and the staging of the tumor is done. If not biopsy induced radiologic artefacts may compromise the findings of imaging.
4. *Heterogenicity:* Malignant sarcomas generaly exhibit heterogenecity. Different types of tissues are seen in different areas. When heterogenecity is suspected multiple samples have to be taken from different areas.
5. *Route of entry:* The entry to the tumor should be from a safe anatomic site avoiding the vital structues such as nerves and vessels.

6. *Hemostasis:* A good hemostasis is a must after biopsy to prevent systemic dissemination of the malignant cells.
7. *Definitive procedure:* Definitive surgical procedure should be planed as early as possible after the biopsy. Frozen section report is reliable enough when it corelates with the clinical and radiological findings and provisional diagnosis.

Techniques of Biopsy

1. *Fine Needle Aspiration Cytology (FNAC):* A 22 guage needle is used. Procedure yields very little tissue. Suitable only when the lesion is homogenous and soft. Rarely used for bone tumors.
2. *Core biopsy:* A 14-gauge needle with a trocar and stilette is used. Core of tissue is taken from multiple sites and studied. Image guidance is always preferred because it helps in placing the needle accurately at the areas to be biopsied.
3. *Open biopsy:* Procedure yields good amount of tissue for histopathologic study. These days, it is employed only when the core biopsy report is inconclusive or ancillary studies are necessary for detailed planning. Open biopsy provides adequate material for performing other supportive studies such as immunohistochemistry, cytogenetics and molecular genetics, etc. These studies are useful in final diagnosis, subclassification of bone tumors, and definitive treatment.

   It is a good practice always to send the biopsied specimen for culture.

Types of periosteal reaction in general

Solid
- Thin
- Thick

Interrupted
- Codman's triangle
- Lamellated (onion skin)
- Perpendicular or Spiculated or Sunray
- Sunburst

Disorganized or complex
- A combination of solid and interrupted.

Laboratory Investigations

Hb%, TC, DC, ESR, serum alkaline phosphatase, serum calcium, serum phosphorus are nonspecific investigations and may help to differentiate tumors from other bone pathologies.

Serum electrophoresis and Bence Jones proteins, help to diagnose multiple myeloma.

Serum acid phosphatase helped in the past to diagnose prostatic carcinoma. These days PSA assay is more widely used.

Biopsy: It is the most useful investigation to establish the diagnosis and grade the tumor.

Management

Total eradication of the lesion is the treatment for bone tumors. In benign tumors, this is possible by surgical excision and reconstruction (when necessary). In malignant tumors, chemotherapy and radiotherapy are necessary in addition to surgical excision and reconstruction. This is to eradicate the tumor from the system and prevent metastasis. Prognosis in those malignant tumors which are radioresistant and chemoresistant is grave, and so is the prognosis when the patient presents late in the course of the disease.

Different applications of chemotherapy

a. *Adjuvant chemotherapy:* It is the therapy which follows primary surgical treatment of malignant tumor. It aims at reducing the risk of recurrence and spread of the tumor.
b. *Neo-adjuvant chemotherapy*: It is the chemotherapy which is administered prior to an ablative procedure. It aims at shrinking the size of the tumor and preventing the spread of tumor during the procedure as well as in the immediate postoperative period.
c. *Radiosensitizing chemotherapy:* It is the chemotherapy given in conjunction with radiotherapy. It aims at sensitizing the malignant cells to the effect of radiation and facilitate destruction.
d. *Curative chemotherapy:* It is the therapy which aims at curing the malignancy.
e. *Palliative chemotherapy:* It aims at providing some comfort to the patient by lessening the symptoms of malignancy.

Radiotherapy: It is generally given by using linear accelerator (Ref: Intensity Modulated Radiation Therapy). It aims at killing the cancer cells using the safe limit of radiation. The damage to the normal tissue is kept to a minimum.

COMMON BENIGN BONE TUMORS

Osteoid Osteoma (Figs 9.2A and B)

This is a tumor consisting of osteoid and woven bone characterized by a central nidus of osteoid surrounded by an exaggerated zone of sclerosis.

- Age: Can occur at any age. Common between 10–30 years. Rare below 5 years and above 45 years.
- Sex: M:F = 2:1.
- Site: It is common in long bones and in the appendicular skeleton. Uncommon in flat bones and axial skeleton. Incidence is about two-third in long bones and one-third in flat bones.
- Size: Approximately 1.5 cm (0.5–2.0).
- Types
  3 types are recognized. They are as follows.
  — Cortical variety which is most common.
  — Cancellous variety which is next common.
  — Subperiosteal variety which is rare.

Clinical Symptoms

Classical focal bone pain which is worse at night and relieved by a small dose of aspirin (this forms the basis of a clinical test i.e. pain relief by aspirin). Focal tenderness may be present. Absence of constitutional symptoms helps to rule out malignancy.

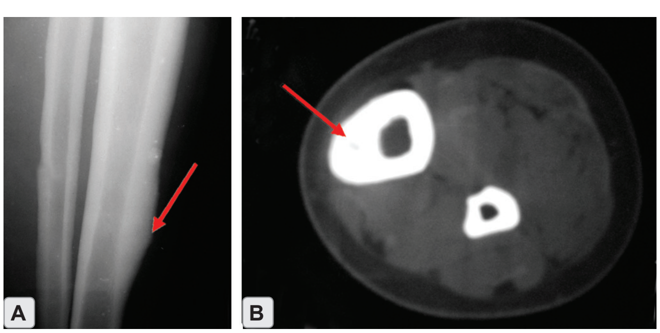
Investigations

Imaging

- *Radiograph:* It shows a central round or ovoid nidus surrounded by an exaggerated zone of sclerosis (Fig. 9.2A).
- *Bone scan by using Technetium 99 m diphosphonate:* It is most sensitive and may show positive results even before radiograph shows signs of the tumor. Also helps in complete removal of the tumor along with the nidus with the help of a hand held radioactive detector.
- *CT scan:* It is useful to localize the lesion precisely. Also useful, in guiding the ablation probe when percutaneous ablation technique is chosen (Fig. 9.2B).
- *SPECT:* Single Photon Emission Computed Tomography is useful in detecting lesions involving spinal arch and spinous process.

Biopsy

- *Histopathology:* Shows a nidus of woven bone rimmed by osteoblasts in a loose fibrovascular tissue along with a reactive thick trabecular bone surrounding the lesion.



Figures 9.2A and B
(A) Radiograph and (B) CT scan showing the nidus of osteoid surrounded by an exaggerated zone of sclerosis.

Treatment

Total excision of the tumor along with nidus gives permanent relief of symptoms.

Cause for pain in osteoid osteoma is said to be due to the presence of axons in the lesion as demonstrated by neural staining techniques as well as marked elevation of levels of Prostaglandin E2.

Benign Osteoblastoma

- Age: Can occur at any age. It is common in younger age group, between 20–30 years.
- Sex: M:F ratio is 3:1.
- Site: Spine. Commonest site being sacrum followed by diaphysis of long bones. Also seen in lower extremities. Rare in flat bones.

Clinical Features

Pain is constant. Not as intense as in osteoid osteoma but long standing and not relieved by salicylates. Painful scoliosis and neurological deficits may develop because of mechanical interference with spinal cord and nerve roots. **Note:** *The neural elements namely the axons, are absent. Hence, pain is not as disabling as in osteoid osteoma. Unlike osteoid osteoma which has limited growth potential, this tumor has a huge growth potential and attains a big size.*

Investigations

The investigations are similar as in osteoid osteoma but the diagnosis is best confirmed by biopsy and histopathological study.

Imaging

- *Radiograph:* shows a variety of features which are not specific. Hence, other imaging techniques are indicated.
- *CT scan:* is done to diagnose a nidus as well as to delineate the margin of the tumor.
- *Bone scan:* only useful to diagnose the site. Not specific.
- *MRI:* useful for determination of soft tissue extension. It only shows non-specific changes of marrow and soft tissue edema.

Biopsy

- *Histopathology:* This tumor shows features that resemble larger version of osteoid osteoma with formation of osteoid and primitive woven bone in a fibrovascular stroma. Hence, the term Giant Osteoid Osteoma is sometimes used to describe this tumor.

Treatment

Total excision and bone grafting is the treatment of choice. Incomplete excision will result in recurrence and the rate is as high as 20%.

There is a variant of Osteoblastoma known as aggressive Osteoblastoma which behaves like an Osteosarcoma in which metastasis and recurrence after excision is very common.

Osteochondroma

This tumor has both osseous and cartilaginous components and hence derives the name Osteochondroma. The cartilaginous portion is generally radioluscent unless it gets calcified.

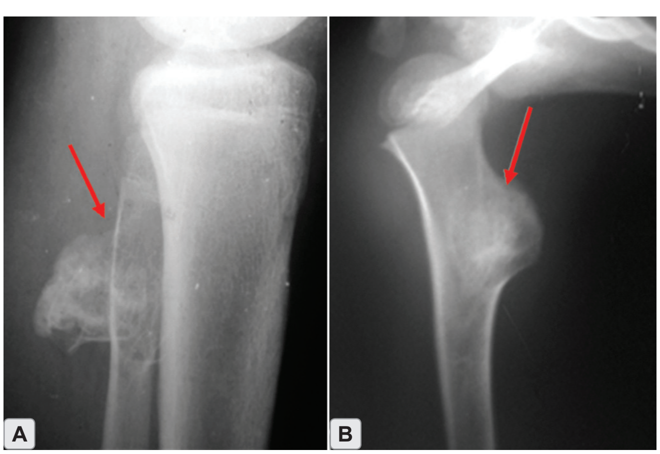
- Age: Common in young adolescent age between 10–20 years. May be present at birth and may manifest in early childhood.
- Sex: M:F ratio 3:1.
- Site: Commonly seen at the epiphysiometaphyseal region (physis), growing away from the physis. Most common site is around the knee. Next common site is around the shoulder.

Types

a. *Pedunculated:* Tumor with a stalk. Commonly seen in long bones and in appendicular skeleton. These are less prone for malignant transformation (Figs 9.3A and 9.4).
b. *Sessile:* Tumor without a stalk. Commonly seen in flat bones and in axial skeleton. These are more prone for malignant transformation (Fig. 9.3B).

Pathogenesis

a. *Theory of herniation of the growth plate:* Trauma or a deficiency of the perichondrial ring may cause herniation of the physis. This herniated physis, continues to function like a physis, producing bone in a manner similar to that of physis. Zones of different stages of bone formation, starting from resting cartilage cell layer to proliferating, hypertrophic and maturing layers are seen.
b. *Theory of genetic abnormality:* This theory is yet to be conclusively proved.



Figures 9.3A and B

(A) Pedunculated and (B) Sessile osteochondroma. Note the age. The presence of growth plate indicates that the patients are adolescents.

Figure 9.4

Classical features of pedunculated osteochondroma. Note the osseous stalk with a bulbous expansion at the end, growing away from the epiphysis.

Clinical Features

The tumor is accidentally detected as an abnormal swelling, which draws attention of an adolescent who is more conscious about his/her cosmesis.

When neglected, it can cause pressure symptoms on neighboring structures namely, the muscles, the vessels and the nerves. It can also cause mechanical obstruction of joint movements. An adventitious bursa can develop followed by recurrent attacks of bursitis. Trauma can cause pathological fracture of the stalk with profuse bleeding. Long standing osteochondromas can transform to chondrosarcomas and present with pain and increase in size of the tumor.

Investigations

Imaging

- *Radiograph:* shows the classical features of osteochondroma. It shows a pedunculated or sessile tumor arising from the epiphysiometaphyseal region and growing away. The cartilaginous cap is not seen unless it gets calcified.

Biopsy

- *Histopathology:* shows various stages of bone formation similar to that seen in the normal physis along with ossified bone at the base of the tumor. Atypical features are seen in the cells only when malignant transformation occurs.

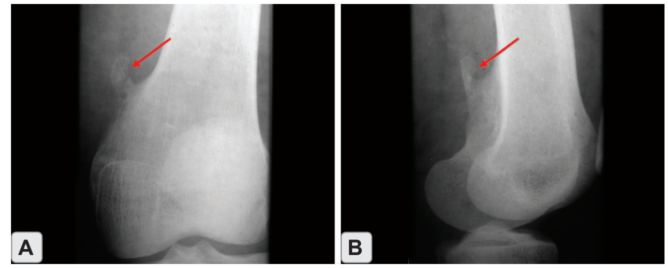
Treatment

Total excision of the tumor along with a good amount of normal bone around the osseous stalk. Recurrence is uncommon after total excision. Ideal period for excision is after skeletal maturity and not before for the fear of recurrence.

Indications for excision

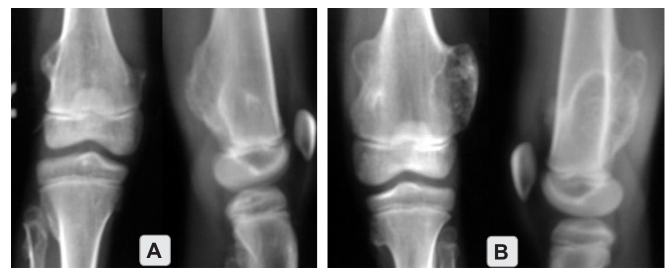
a. Cosmetic reasons
b. Recurrent attacks of bursitis
c. Pressure symptoms
d. Mechanical obstruction
e. Fracture of the stalk (Figs 9.5A and B)
f. Malignant transformation

Osteochondromatosis: It is also known as Heriditary Multiple Exostosis, Metaphyseal or Diaphyseal Aclasis (meaning of the word Aclasis is, a pathological outgrowth). First identified by John Hunter (1786). It is an autosomal dominant disorder running in families.



Figures 9.5A and B

A **rare complication of fracture of the stalk** of an osteochondroma. The incident occurred during a soccer match, when the ball hit the tumor. Until then, patient did not have any symptoms.



Figures 9.6A and B

Metaphyseal Aclasis. Note the presence of multiple sessile osteochondromas arising from the metaphysis of the femur and the tibia.

M:F ratio 1:1 and detected as early as 3–4 years of age. It affects multiple bones which results in growth abnormalities and deformities. Malignant transformation is also common when compared to Solitary Osteochondroma (Figs 9.6A and B).

Dysplasia Epihysialis Hemimelica (DEH): It is also known as Trevor's disease and is characterized by osteochondromas occurring in one limb and on one side of the epiphysis. Medial side is twice commonly affected than the lateral side.

Chondroblastoma

This tumor was first identified by Codman in the year 1931 who termed it as 'Chondroblastic variant of Giant cell tumor'. Henry Jaffe and Louis Lichtenstein a decade later gave the name 'Benign Chondroblastoma', signifying the chondroblastic origin of this tumor. It accounts for about 1% of all bone tumors. Though considered as benign, local erosion and distant metastasis to lungs is seen at times.

- Age: 10–30 years.
- Sex: M:F ratio 3:1.
- Site: Epiphysis of a long bone. Common site is around the knee. Next common site is proximal femur and proximal humerus. Rarely seen in pelvis, talus, calcaneum and patella.

Clinical Features

Slow growing tumor. It may remain asymptomatic for quite a long period. Presents with swelling and pain. Joint effusion may be present. It may also present with a pathological fracture.

Investigations

Imaging

- *Radiograph:* Shows a lytic area in the epiphyseal region extending into the metaphysis. Speckled calcification may be seen.
- *CT:* Delineates the tumor better and shows calcified areas.
- *MRI:* Helps in identifying transepiphyseal and transcortical extension of the tumor.

Biopsy

- *Histopathology:* The tumor consists of primitive chondroblasts in a dense eosinophyllic matrix. Coarse calcification may be seen (Chicken-wire calcification). Atypism and mitotic figures are not seen unless malignant transformation has occurred.

Treatment

Total excision of the tumor with bone grafting to fill the defect is the treatment of choice. Incomplete excision may lead to recurrence of the tumor. Recurrence rate is as high as 10–35%.

Note: *Since many features are common to both Giant cell tumor and benign Chondroblastoma, it is considered as one of the differential diagnosis for Giant cell tumor. Presence of 'speckled calcification' radiologically and 'chicken-wire' calcification histopathologically, goes in favor of benign Chondroblastoma.*

Chondromyxoid Fibroma

This is a tumor comprising of chondroid, myxoid and fibrous tissue elements and hence derives its name. It was first described by Jaffe and Lichtenstein, in 1948. It accounts for about 1% of bone tumors.

- Age: 15–30 years.
- Sex: M:F ratio 1:1 or a little more in men.
- Site: In the metaphysis of a long bone extending on to the epiphysis and the diaphysis. Common site is the upper end of tibia followed by lower end of the femur and the upper end of humerus.

Clinical Features

Many a times it is discovered accidentally. Often it is not diagnosed until the tumor grows to a considerable size or causes pain. Limitation of joint movement is not seen.

Investigations

Imaging

- *Radiograph:* Shows an expansile, eccentric, lytic, metaphyseal lesion with a regular or at times irregular sclerotic margin.
- *CT and MRI:* help in better evaluation and planning the surgery.

Biopsy

- *Histopathology:* The tumor is supposed to arise from cartilage forming connective tissue of the marrow. It comprises of chondroid, myxoid and fibrous tissue. No atypism is seen. Malignant transformation is rare and when occurs it is difficult to distinguish from Primary Chondrosarcoma.

Treatment

Total excision and bone grafting is the treatment of choice.

Enchondroma

It is a benign, solitary, intramedullary, cartilaginous neoplasm.

- Age: 20–40 years.
- Sex: M:F ratio 1:1.
- Site: Common in small bones of the hand and feet. Next common sites are long bones and pelvis. The location is diaphyseal in short tubular bones and metaphyseal in long tubular bones.

Clinical Features

Early: Swelling is the only feature which draws attention.

Late: Swelling associated with pain due to

a. Pathological fracture.
b. Malignant transformation.

Note: *Pathological fracture more commonly occurs when malignant transformation takes place.*

Pathogenesis

The cartilaginous cell rests, get displaced during growth and lie in the metaphysis/diaphysis. They start

proliferating and replace the normal bone by mineralized or nonmineralized hyaline cartilage.

Investigations

Imaging

- *Radiograph:* Shows a solitary lytic lesion in the bone involved.
- *CT scan:* Helps to diagnose subtle calcification.
- *MRI:* Helps to differentiate bone infarct from enchondroma.

Biopsy

- *Histopathology:* Seen under microscope as lobules of cartilage containing chondrocytes surrounded by a narrow rim of bone. Calcification may or may not be present.

Treatment

Excision of the tumor and bone grafting (care should be taken to prevent spillage of tumor at the donor site of bone graft because the tumor can develop by seeding).

Chondrosarcoma is a likely complication of enchondroma. Hence, it is important to have a periodic follow-up when surgery is deferred.

Syndromes associated with enchondromas are:

a. Ollier disease is nonhereditary and presents with multiple enchondromas.
b. Mafucci's syndrome is nonhereditary and presents with multiple enchondromas and multiple hemangiomas.
c Metachondromatosis is a hereditary disorder, autosomal dominant and presents with multiple enchondromas and osteochondromas.

Aneurysmal Bone Cyst (ABC)

This is an expansile, destructive (osteolytic) lesion, consisting of blood filled spaces separated by trabeculae of osteoid or septae, with interspersed osteoclasts. It was first described by Jaffe and Lichtenstein in the year 1942. It accounts for about 6% of bone tumors.

- Age: 10–20 years.
- Sex: M:F ratio 1:1.5. Incidence is slightly more in females.
- Site: In the metaphysis of long bones. Sometimes occurs subperiosteally. Rare in the spine and flat bones.

Etiology

The etiology is unknown and thought to be multifactorial. Two likely causes are:

a. Because of AV malformation and altered circulatory function.
b. Degeneration and destruction occurring secondary to trauma and formation of hematoma.

Clinical Features

Pain, swelling and at times a pathological fracture. Considerable time may lapse before the symptoms develop. Involvement of the spine may cause neurologic symptoms.

Investigations

Imaging

- *Radiograph:* Classical cases may show 'Finger in the balloon' sign radiologically.
- *CT, MRI:* Pictures vary depending on the phase of the cyst.

  Four distinct phases are recognized.

  a. Osteolytic phase.
  b. Active phase of rapid destruction.
  c. Maturing phase/phase of stabilization.
  d. Healing phase.

  The osteolytic phase presents as a focal area of osteolysis.

  The active phase presents with occurrence of rapid destruction, thinning of the cortex and bulging of the periosteum (periosteal blow out).

  The maturing phase presents with formation of distinct peripheral bony shell and internal septae (this gives rise to a 'soap bubble' appearance).

  The healing phase presents with irregular new bone formation as a result of progressive calcification and ossification.

Biopsy

- *Histopathology:* Thin walled blood filled cavities along with trabeculae of osteoid or septae with interspersed osteoclasts.

Treatment

a. Surgical excision and bone grafting is the treatment of choice whenever feasible.
b. Arterial embolization has shown successful results in selected cases (e.g. when there is extensive subchondral involvement which may compromise joint function after extensive surgical excision or in areas which are surgically inaccessible for a complete excision).
c. Intralesional injections like phenol, liquid nitrogen, etc. have not gained popularity. Their results are uncertain and there is a great risk of injury to neighboring structures like vessels and nerves.

Unicameral Bone Cyst (UBC)

Is a unique benign cystic lesion filled with fluid seen in children. Virchow, recognized this lesion as early as 1870. In 1942, Henry Jaffe and Louis Lichtenstein published their article about unicameral bone cyst (Fig. 9.7).

- Age: 5–15 years.
- Sex: M:F ratio 2:1.
- Site: Proximal humerus followed by proximal femur (account for about 90%).

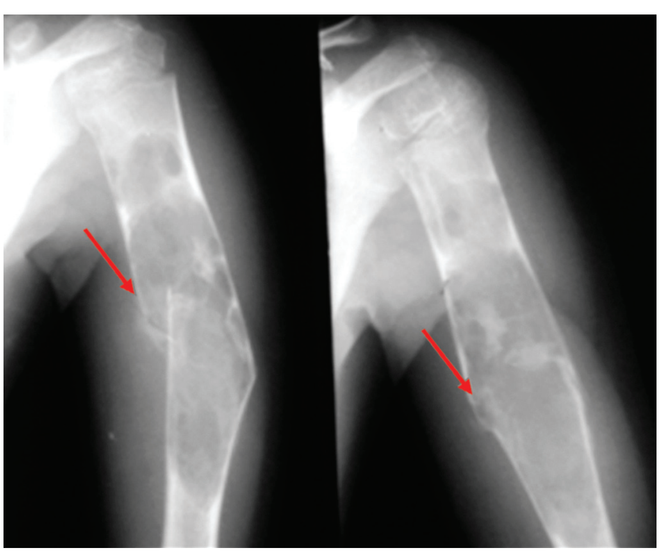


Figure 9.7

A huge unicameral bone cyst with classical features of pathological fracture, loculations and extension into the diaphysis.

Etiology

Several theories have been put forth:

a. Blockage in drainage of interstitial fluid in rapidly growing and rapidly remodeling cancellous bone—Cohen 1960.
b. Congenital cell rest of synovial tissue as proved by the presence of macrophage like synovial type A and fibroblast like type B cells in the lining of the cyst—suggested by Joseph M Mirra.

Clinical Features

Always detected accidentally, except in cases with pathological fracture where patient presents with pain. When the cyst is adjacent to growth plates, it is considered as an active cyst and when at a distance, it is considered as a latent cyst.

Investigation

Imaging

- *Radiograph:* Shows an osteolytic lesion in the metaphysis extending to the diaphysis. At times may show loculations. Evidence of pathological fracture may be present.

  The fallen fragment sign—this sign is identified by the presence of a bone fragment in the dependent portion of a cystic bone lesion and is said to be diagnostic for a simple (unicameral) bone cyst, particularly following a pathological fracture.

  This occurs because a simple bone cyst is fluid filled, and therefore a bony fragment can descend by gravity through the fluid, uninterrupted. In contrast, the other differential lesions such as fibrous dysplasia, aneurysmal bone cyst and enchondroma, seen as lytic tumors radiologically, do not have such fluid filled spaces. Therefore, they would not permit such a movement of the fragment.

Biopsy

- *Histopathology:* Shows empty space filled with fluid and the wall of the cavity is lined by a thin layer of connective tissue.

Treatment

As spontaneous resolution is seen, asymptomatic cysts when detected accidentally are left alone with periodic follow-up till adulthood. Treatment is necessary only if no resorption is observed. Pathological fractures are treated nonoperatively by immobilization. Healing invariably occurs.

Percutaneous techniques

a. Steroid in the form of methylprednisolone is injected into the cyst. Results have been satisfactory.
b. Bone marrow injection with about 25 ml of autologous marrow aspirated form the iliac crest and injected immediately into the site. Resolution has been observed.

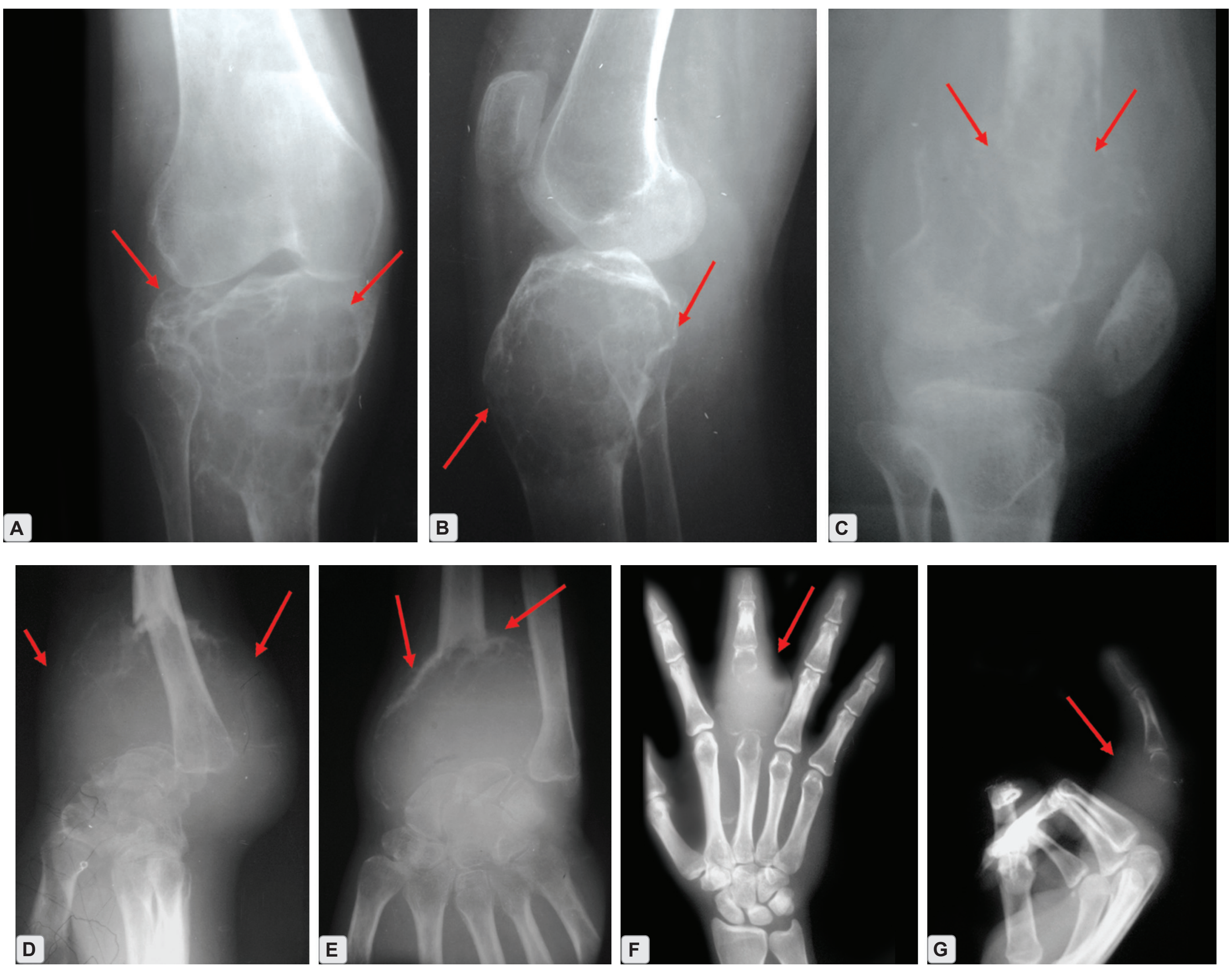
These procedures are yet to be accepted universally.

Operative procedures: Total excision and bone grafting done only selectively. It is a highly morbid procedure for young individuals. Hence, to be viewed critically.

COMMON MALIGNANT BONE TUMORS

Giant Cell Tumor (GCT) (Figs 9.8A to G)

It is considered as a locally malignant tumor of the bone because local recurrence is more common (around 50%) when compared to distant metastasis (occurs only in 5–10% of the cases). It is said to have been first reported by Cooper in 18th century. It accounts for about 5% of bone tumors.



Figures 9.8A to G

GCT arising from various bones. Note the epiphyseal, expansile, eccentric and lytic nature of the lesion. 'Soap bubble' appearance is seen here in tibial GCT (A and B). All the cases seen here are advanced and late presentations with pathological fractures.

- Age: 20–40 years.
- Site: Epiphyseal ends of the long bones. Upper end of tibia, lower end of the femur, lower end of radius and proximal humerus are the common sites of involvement in the order of frequency. Rare in the vertebra and other spongy bones like calcaneus, talus and pelvis.
- Sex: A little higher predilection for female sex is seen.

Clinical Features

Presents as a slow growing, painful swelling arising from the epiphyseal end of a long bone. Egg shell crackling may be present on palpation when thinning of the cortex and pathological fracture has occurred. Limitation of joint movement is a late feature.

Investigations

Imaging

- *Radiograph:* Shows classical epiphyseal, eccentric, expansile, lytic lesion which is loculated (at an earlier age giving a soap bubble appearance) or non-loculated (seen in a mature skeleton after growth plate completely closes).

  Cortex is thinned out. Pathological fracture and invasion into a joint is a late feature. When seen early in the course of the disease, it points out towards the aggressiveness of the tumor.
- *CT; MRI:* helps in better delineation of the extent of involvement.

Biopsy

- *Histopathology:* A picture consisting of spindle shaped and rounded mononuclear cells with osteoclastic type multinuclear giant cells and blood vessels is seen under microscope. Jaffe (1940) took into consideration the aggressiveness of the tumor and graded it into three grades. The Netherlands committee on bone tumors graded it into four grades. While grading, atypism shown by the stromal spindle shaped cells alone is taken into consideration whereas features shown by multinucleated giant cells are ignored. The more the atypism, i.e. anisocytosis, anisonucleosis, hyperchromatism, etc., the more aggressive (malignant) is the tumor. The number and size of the multinucleated giant cells proportionately gets reduced as the atypism of stromal cell increases. This grading is questionable in today's practice.

Based on Jaffe's Grading (1940)

Grade I: No/Minimal atypism of stromal cells. Giant cells are many and big sized. Grade II: Moderate atypism of stromal cells. Giant cells are a few and big sized.	Benign
Grade III—Severe atypism of stromal cells. Giant cells are a few and small sized.	Borderline malignant

The Netherlands committee on bone tumors has added one more grade.

Grade IV: Sarcomatous dedifferentiation is observed. Very few small sized giant cells (resembling malignant fibrous histiocytoma).	Malignant

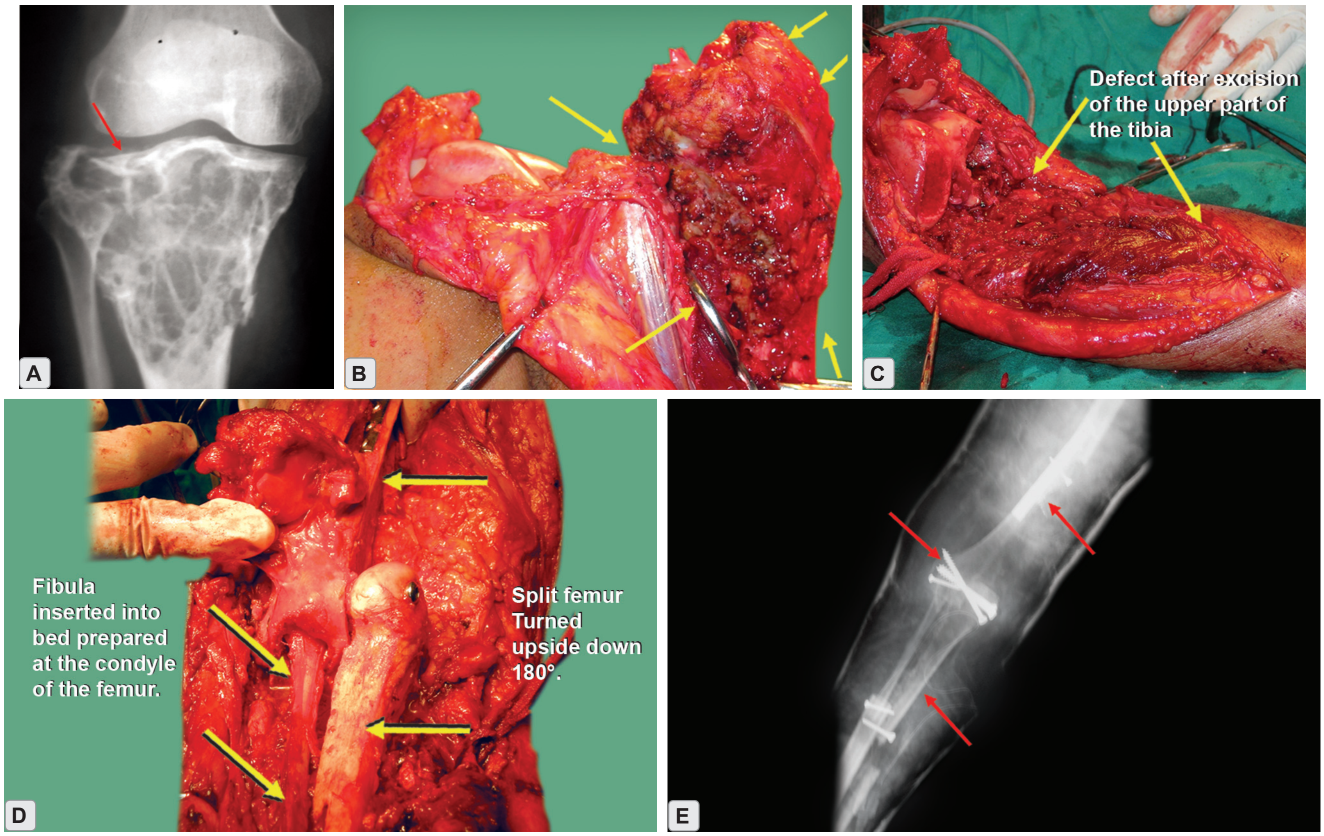
Treatment

Total excision and reconstruction (Figs 9.9A to E) either by filling the defect with bone grafts, polymethyl methacrylate or both is the usual treatment. When reconstruction and salvage of the joint is not possible, arthrodesis/total joint replacement with custom made prosthesis is to be considered.

Solitary pulmonary metastasis when detected has to be excised locally.

Radiotherapy is reserved as a palliative procedure for tumors that arise from surgically inaccessible sites.

Prognosis: Good in Grade I and II, when totally excised. Local recurrence is common than distant metastasis.



Figures 9.9A to E

(A) A large GCT with pathological fractures and with indications that it has come out of its compartment (extra compartmental). (B to D) are the steps of the procedure of 'Turn plasty' showing total excision of the upper part of the tibia along with the tumor and reconstruction by turning one half of the lower femur and attaching it to the remaining tibia (E) Radiograph taken after the 'Turn plasty' procedure is complete.

Osteosarcoma

This is a highly malignant intramedullary bone tumor with high rate of mortality. It accounts for about 35% of bone malignancies.

The term 'Osteosarcoma' was coined in the year 1805 by Alexis Boyer, personal surgeon to Napoleon.

Types

Primary:

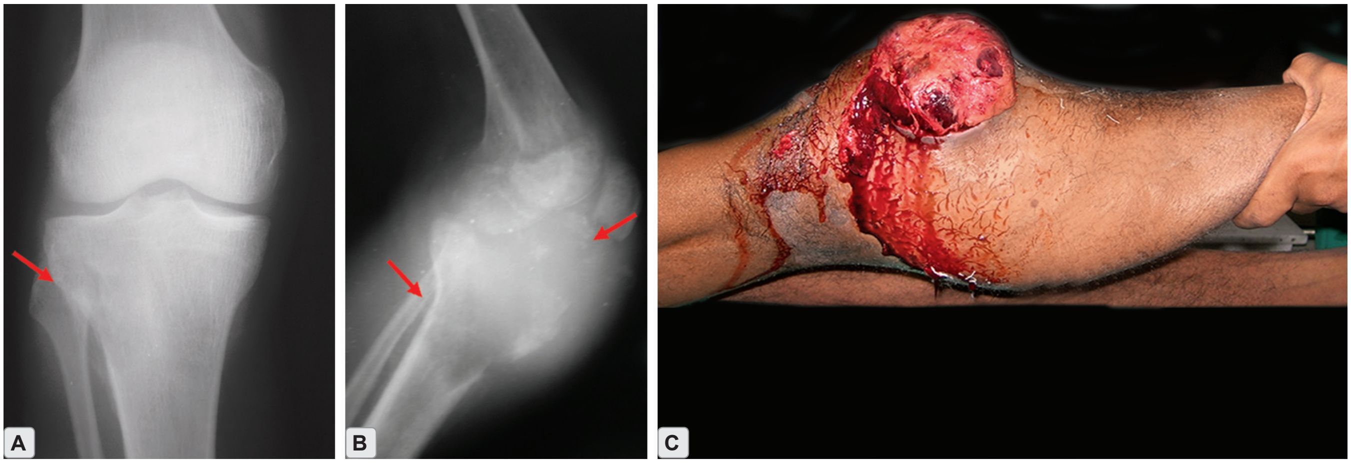
a. Osteoblastic b. Chondroblastic c. Fibroblastic d. Sclerosing e. Osteolytic f. Telangiectatic (Figs 9.10A to C). g. Multifocal h. Parosteal i. Periosteal	These are the conventional types and their names are given depending on the predominant tissue (a to f) as well as location (g to i)

Secondary: e.g. Osteosarcoma developing in Paget's disease, fibrous dysplasia, etc.

- Age: 10–20 years (except in secondary osteosarcoma which is seen in older age group).
- Sex: Incidence is slightly more in males.
- Site: Metaphysis of long bones. Common in lower end of femur, upper end of tibia and upper end of humerus. It is rare in jaw and skull. Very rare in extra skeletal sites. Multifocal origin is also seen rarely (either synchronously or metachronously).

Clinical Features

Pain is the earliest presenting feature which becomes a cause for concern. The pain gets aggravated with activity but not relieved by rest. Gradually, limp/limitation of joint movement develops as swelling appears. It may be associated with constitutional symptoms.



Figures 9.10A to C

A case of telangiectatic osteosarcoma. (A) At early presentation when grave nature of the tumor was explained and surgery was advised. Patient refused surgery and went for native treatment. (B and C) After 6 months of refusal of treatment and taking native medicine. Note the rapid increase in size, extensive destruction without new bone formation seen radiographically and fungation with active bleeding seen clinically.

Investigations

Imaging

- *Radiograph:* In typical cases, 'Sun ray' or 'Sun burst' appearance with Codman's triangle is seen. Cumulus cloud pattern, in Sclerosing variety and expansile, eccentric lysis, in telangiectatic variety are the other radiographic features.
  Sun burst or Sun ray appearance is seen because of bone formation along the Sharpey's fibers which firmly bind the periosteum to the bone (Figs 9.11A and B).
- *Bone scan, CT, MRI and PET CT*: are useful in detailed evaluation and planning the required treatment.

Biopsy

- *Histopathology:* This tumor is thought to arise from the primitive mesenchymal cells and is diagnosed by the presence of malignant stromal cells laying down tumor osteoid (osteoblastic), along with areas of hemorrhage and necrosis along with spicules of destroyed bone. Depending on the predominant tissue it can be classified as chondroblastic, fibroblastic, telangiectatic, etc.

Treatment

Primary objective of treatment is to have a long-term disease free survival. Limb salvaging is of secondary importance. Thus, the treatment aims at increasing the survival rate with restoration of maximum function when feasible. **Limb salvage should always be attempted when possible but one should not hesitate to recommend amputation when needed.**

The approach is multidisciplinary. It varies depending on the site, size, type, local extension and distant spread. Preoperative chemotherapy has increased the 5 years survival rate from 12% to 65–75%. Preoperative radiotherapy is given only when metastasis is detected.

Enneking system of staging (1980) helps in planning of surgery and chemotherapy(Refer beginning of this chapter):

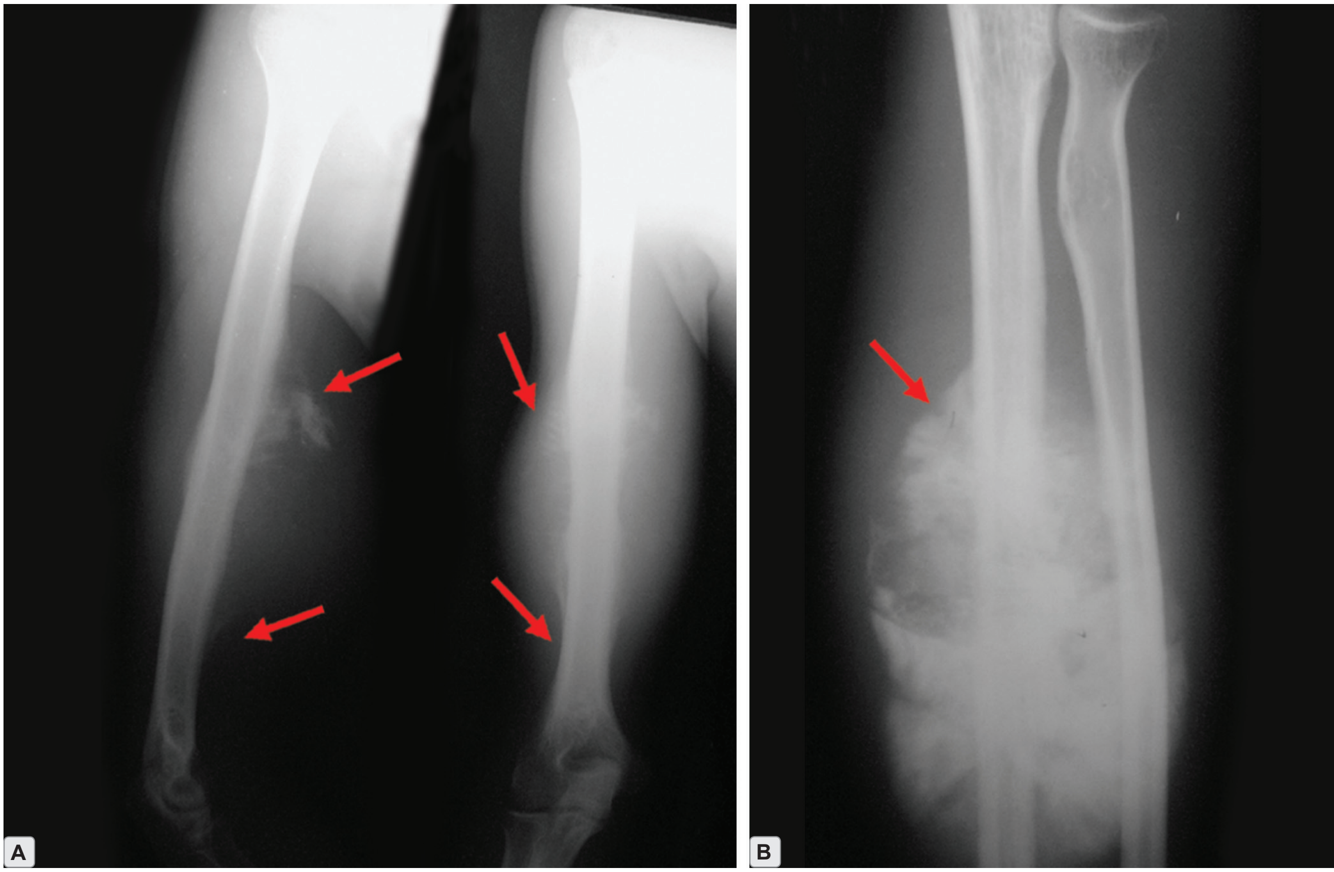
Preoperative chemotherapy (Neoadjuvant chemotherapy): Is useful and effective in shrinking or circumscribing the tumor mass and of help in planning the surgical procedure, i.e. feasibility of a limb salvaging procedure. It also can prevent the spread of the disease.

Drugs used are methotrexate, adriamycin and cisplatin (MAP regimen), in a cyclical manner. Expert evaluation is individually necessary to assess the physiological limit of tolerance of these cytotoxic drugs. Careful monitoring of heart, lungs, kidney, liver and hemopoietic system is a must.

Poor response: If the response is poor, other drugs like ifosfamide or etoposide are added.

Good response: If the response is good, interferon is given in an attempt to get a cure.

Five years survival rate is less in those who show poor response and vice versa.



Figures 9.11A and B
Radiograph showing features of osteosarcoma. Note the Codman's triangle at the lower humerus in AP view, indicating reactive new bone formation.

Angiography

a. Helps to localize the displaced vessels.
b. Tumor blush: Osteosarcoma is a tumor which promotes neovascularization. Hence, there is enhancement of the contrast medium. This is referred to as 'Tumor blush'. After giving chemotherapy if this neovascularization is not seen (disappearance of vascularity) it infers that the chemotherapy has been effective.

Surgical Procedures

Surgical procedures in Osteosarcoma fall into two categories

a. Limb salvaging
   Limb salvaging procedures when feasible should always be preferred especially in early stage of the disease and when the tumor is localized. Enneking system of staging helps in taking a decision and planning the treatment (Refer beginning of the chapter).
   These procedures aim at radical excision of the tumor and reconstruction with autograft (fibula)/allograft/custom made prosthesis. Neoadjuvant chemotherapy is a must before proceeding with these procedures for preventing seeding, spread and development of metastases. Chemotherapy should be continued after the surgery.
b. Life saving
   These are amputations. They are preferred only in advanced cases where limb salvaging is impossible and are done in an attempt to save the life of the patient. Adjuvant/Palliative chemotherapy is always given after amputations.
   Other procedure:
   Rotation plasty: This is a procedure described in lower limbs where, an en mass resection of tumor along with distal femur and proximal tibia is done. Then, the distal portion of the limb along with remaining distal tibia is turned to 180°, drawn

proximally and fixed to the proximal femur, so that the ankle becomes the knee. A suitable prosthesis gives considerable function. The procedure is always followed by adjuvant/palliative chemotherapy.
Lung metastases are treated by local resection.
Radiotherapy, when indicated has to be given.

Chondrosarcoma

Chondrosarcomas are the next common sarcomas seen after osteosarcomas and account for about 25% of the primary malignant skeletal noeplasms. They present with a highly varied behavior from slow growing non-metastasizing tumor to fast growing metastasizing tumor. Histopathological features too vary accordingly.

- Types: Primary and secondary
  Central (intramedullary) and peripheral
- Age: Primary occurs above 40 years
  Secondary occurs 20–40 years
- Sex: M:F ratio 2:1
- Site: Flat bones like pelvis and scapula are the common sites. They also arise from the metaphysis of long bones like femur, tibia and humerus.

Clinical Features

Presents with dull aching pain in the vicinity of a joint, aggravated by movement, worse at night. Joint effusion and limitation of movements develop later when swelling starts appearing.

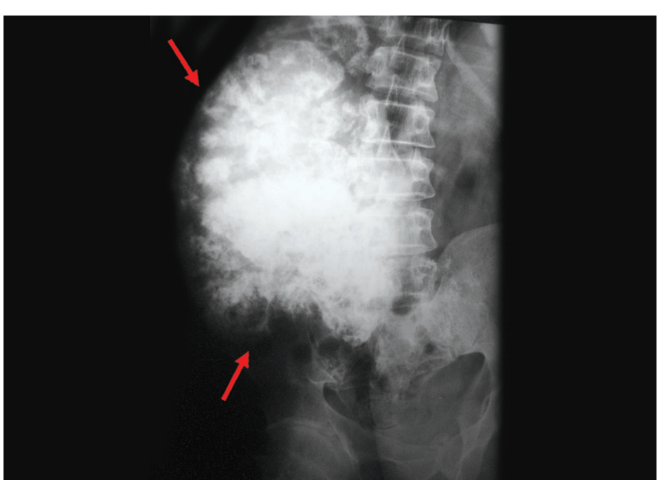


Figure 9.12

A huge secondary chondrosarcoma arising from an aclatic lesion in the ilium in a patient with metaphyseal aclasis. Being radioresistant and chemoresistant and in this instance inoperable, it proved fatal.

Investigations

Imaging

- *Radiograph:* shows an expansile tumor with speckled calcification (Fig. 9.12).
- *CT, MRI and PET CT*: help in further delineation and evaluation of spread of the tumor.

Biopsy

- *Histopathology:* It presents a varied picture of a well-differentiated tumor producing normal hyaline cartilage to poorly differentiated tumor producing atypical hyaline cartilage. Hence grading is done. Grade I—low grade malignant; Grade II—moderately malignant; Grade III—high grade malignant. Calcification may be seen in some areas. In addition, three variants are recognized histopathologically:
  a. *Mesenchymal chondrosarcoma:* Presents with bimorphic picture of both low and high grade malignancy. Common in spine, ribs and jaw.
  b. *Clear cell chondrosarcoma:* It is a low grade chondrosarcoma consisting of clear cells with vacuolated cytoplasm. Matrix is significantly calcified. It is common in the epiphysis of the femur and the humerus.
  c. *Dedifferentiated chondrosarcoma:* It is a highly malignant tumor presenting with features of other sarcomas like osteosarcoma, malignant fibrous histiocytoma, etc.

  The 5-year survival in Grade I lesions is around 90% and in Grade III lesions is around 30%.

Treatment

Wide surgical excision is the treatment of choice. Local recurrence is common by seedings than distant metastasis except in aggressive type where pulmonary spread occurs via bloodstream. Chondrosarcomas are resistant to radiotherapy and chemotherapy. Hence, there is no role for radiotherapy and chemotherapy.

Ewing's Tumor

Described by James Ewing in the year 1921. Hence, derives the name. It is a highly malignant primary bone tumor derived from the red marrow. It accounts for about 33% of primary bone tumors and stands second among the common malignant bone tumors in the young.

- Types
  a. Sclerotic
  b. Lytic.
- Age: 5–15 years. Rare before 5 years and after 30 years.
- Sex: M:F ratio 1.5:1
- Site: Diaphysis of a long bone extending into the metaphysis. It is rare in flat bones.

Clinical Features

Presenting feature is pain of insidious onset which is intermittent initially and then progresses to become continuous and more severe. Next, a rapidly growing swelling develops. If the axial skeleton is involved, neurological deficits may be observed. Constitutional symptoms such as fever, malaise, etc, may be present.

Investigations

Imaging

- *Radiograph:* shows a lytic, permeative lesion not well demarcated. The pattern of bone destruction varies from permeative pin head sized holes, moth eaten, rotten wood to pure lytic. Hence, it can mimic a variety of bone tumors, both benign and malignant. Periosteal reaction manifests with formation of a typical 'onion skin' appearance (Fig. 9.13) and is identified as a late and advanced feature of the tumor. Codman's triangle may be present.

Figure 9.13

Layers of new bone formation giving rise to 'onion skin' appearance. Note that the tumor is primarily intramedullary, diaphyseal and extending into the metaphysis.

- *CT; MRI; PET CT:* help in better delineation and evaluation of spread of the tumor.

Biopsy

- *Histopathology:* Highly cellular tumor comprising of sheets of small round neoplastic cells with large oval nuclei. Exhibits hyperchromatism (stippled chromatin) and mitotic figures. Cytoplasm is sparse with ill defined cytoplasmic membrane. Pseudo-rosette formation may be present.
  Note: *When suspected following additional investigations are done for confirmation.*
  a. Immunohistochemisrty
  b. Cytogenetics
  c. Electron microscopy

Treatment

As occult metastasis is very common in these tumors, multidrug chemotherapy and local control by means of radiotherapy or surgery (only if local lesion is resectable) is the treatment of choice. This improves the chances of survival. Thus, the approach is a multidisciplinary one.

The widely followed regimen is VAdriaC regimen/ (VAC) i.e. vincristin, adriamycin and cyclophosphamide. Ifosfamide and etoposide are used when indicated to improve the outcome. After this multidisciplinary approach the 5-year survival rate has risen from 10% to almost 70%.

Myeloma

Is a tumor arising from neoplastic proliferation of plasma cells or myeloma cells. Depending on the site and nature of proliferation, several presentations are recognized.

Types

Multiple myeloma: Presents with multiple skeletal foci of proliferation (about 50%).

Solitary myeloma: Is also known as plasmacytoma, presents with a solitary skeletal foci of proliferation (about 25%).

Myelomatosis: Also known as generalized myeloma, presents with a generalized and diffuse involvement of the skeleton. Results in thinning of the skeleton, resembling osteoporosis (about 15%).

Plasma cell leukemia: Malignant plasma cells in abundance in the bloodstream (about 1%) is rare.

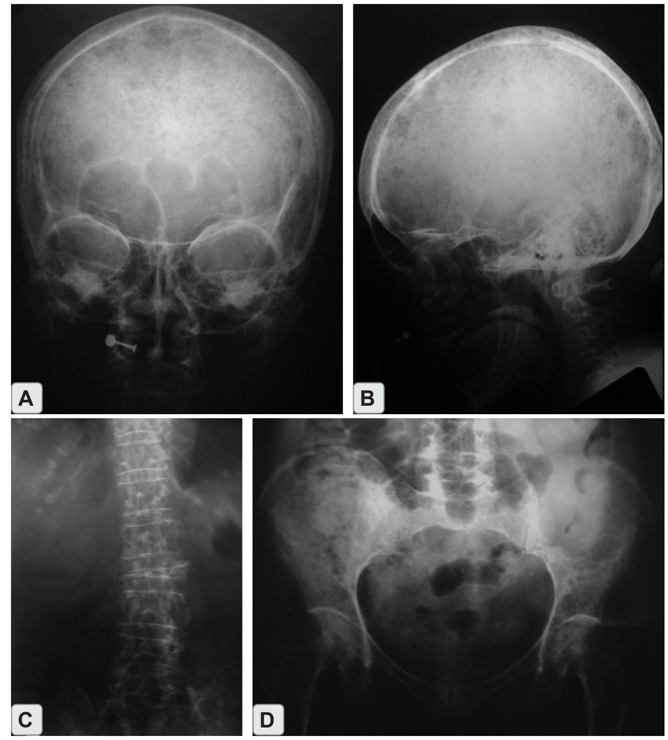
Extraskeletal myeloma: Very rare. It is a variant and presents with a solitary tumor in the nasopharyngeal or oral cavity.

- Age:
  - Multiple myeloma 60–70 years.
  - Solitary myeloma 50–60 years.
- Sex: M:F ratio is 3:1
- Site: Originates from the bones containing red marrow. Hence, multiple myeloma is common in axial skeleton and flat bones than appendicular skeleton and long bones.

  Solitary myeloma is common in the diaphysis of long bones and in the vertebral body.

Clinical Features

It presents with aching type of pain which is intermittent to begin with. It is either generalized or localized and relieved by bed rest. Later, pain becomes more severe and constant. Constitutional symptoms develop. Easy fatigue is seen due to ensuing anemia and bleeding tendency. Recurrent infections may develop because of the replacement of marrow by tumor cells. Renal failure (Myeloma kidney) and pathological fracture can develop late in the disease.



Figures 9.14A to D

Generalized punched out lesions in the skull, vertebrae, ribs and pelvic bones which are classical of multiple myeloma.

Investigations

General

a. Hb%, TC, DC, ESR

   Low Hb%, low TC, granulocytopenia, raised ESR

b. Platelet count

   Thrombocytopenia

c. Serum calcium, phosphorus, alkaline phosphatase

   Raised serum Ca, ALP is normal except in patients with pathological fracture.

d. Electrophoresis for increased gamma globulin fraction.

   Shows increase in gamma globulin fraction.

e. Urine for 'Bence Jones' proteins.

   Described by English physician Henry Bence Jones in the year 1847.

   These are immunoglobulins produced by plasma cells. These light chain molecules, precipitate at 60°C and disappear on further heating at 90°C.

   Reappear on cooling to 60°C.

f. Bone marrow aspiration biopsy.

   This is confirmatory. It shows, characteristic plasma cells—myeloma cells.

Imaging

- *Radiograph:* shows solitary or multiple punched out lesions or generalized osteopenia with or without pathological fracture/s (depending on presentation) (Figs 9.14A to D).
- *CT; MRI; PET CT*: help in detailed evaluation with respect to nature of the tumor as well as spread of the disease, and in instituting the required therapy.

Biopsy

- *Histopathology:* Nodular or diffuse infiltrate of plasma cells with loss of marrow fat and dissolution of bone is observed. These cells have a single large 'clock face' or 'cart wheel' nucleus with abundant basophilic cytoplasm along with rich endoplasmic reticulum. Perinuclear halo is characteristic. Atypism with presence of two or more nuclei, loss of perinuclear halo, loss of clock face and presence of mitotic figures, indicate malignancy.

Treatment

- Stable (asymptomatic) otherwise known as 'smoldering myeloma' does not require treatment.
- Unstable (symptomatic) myeloma is treated by chemotherapy, radiotherapy and bone marrow transplantation. Treatment aims at replacing the abnormal marrow with normal marrow by means of peripheral stem cell transplantation. This may result in complete remission.
- Some of the chemotherapeutic regimens are as follows:
    - i. Thalidomide/dexamethasone, vincristine
    - ii. Vincristine, adriamycin, dexamethasone
    - iii. Melphalan, prednisone
    - iv. Melphalan, prednisone, thalidomide
    - v. Cyclophosphamide, prednisone

METASTATIC BONE DISEASE

These are generally metastasis from adenocarcinomas. Following adenocarcinomas are known to cause secondaries in the bone (Fig. 9.15).

i. Thyroid
ii. Breast
iii. Lungs
iv. Liver

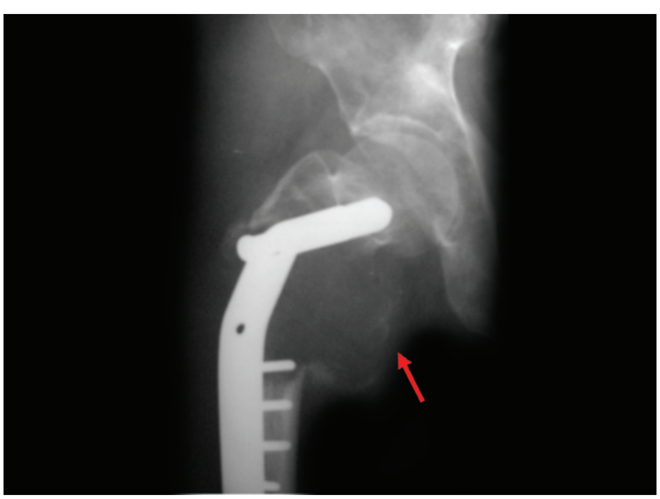


Figure 9.15

Radiograph of a missed diagnosis. A pathological fracture which was routinely fixed as a traumatic fracture. Patient was not relieved of pain even after surgery. An X-ray taken after 2 months confirms the diagnosis of a secondary.

v. Kidneys
vi. Prostate.

Of these, all are lytic in nature except from the prostate which is sclerotic. At times, the primary tumor is not detectable with routine investigations and bone scan along with PET CT are useful for identification and assess the extent of spread of the disease.

The treatment for metastatic bone disease is always palliative and aims at providing pain relief and increasing the comfort in a terminally ill patient.

A summary of different radiological features seen in bone lesions and their probable diagnosis

Radiological features	*Probable diagnosis*
"Kissing" bones (lytic lesions in adjacent epiphysis)	GCT, angiosarcoma, pigmented villonodular synovitis, infections
Codman's triangle	Osteosarcoma, osteomyelitis, ABC
Complete sclerotic rim, no break	Benign lesion (95% accuracy)
Cumulus cloud appearence	Osteosarcoma, stress fracture
Epiphyseal, solitary, eccentric lytic lesion with sclerotic margin	Chondroblastoma, enchondroma, GCT
Epiphyseal, solitary, lytic lesion without sclerotic margin	GCT
Expansile lesion, poorly demarcated with windblown calcifications	Chondrosarcoma
Expansile lesion nontrabeculated lesion	Benign tumor (majority of cases), grade I sarcoma, solitary myeloma, metastasis (a small percent of cases)
Expansile, trabeculated lesion	Grade I sarcoma, GCT, myeloma
Fallen fragment sign	Simple bone cyst
Finger-in-the-balloon appearance	ABC
Ground glass appearance	Fibrous dysplasia, osteoblastoma, grade I osteosarcoma
Onion-skinning	Ewing's sarcoma, subacute osteomyelitis, eosinophilic granuloma
Ring-like to popcorn density	Enchondroma and secondary chondrosarcoma

Revision Questions

Q. Write notes on
a. Osteoid osteoma
b. Enchondroma
c. Benign chondroblastoma
d. Aneurysmal bone cyst
e. Unicameral bone cyst
f. Giant cell tumor
g. Osteochondroma
h. Chondromyxoid fibroma
i. Osteosarcoma
j. Chondrosarcoma

Q. Write briefly on
a. Multiple myeloma
b. Myelomatosis
c. Bence Jones proteins
d. Electrophoresis
e. Plasma cells

Q. Write notes on
a. Codman's triangle
b. Sun ray appearance
c. Onion skin appearance
d. Metastatic bone disease
e. Pathological fracture
f. PET CT
g. MRI
h. Bone scan

Essay Questions

Q. Discuss the clinical features, radiological features, diagnosis and management of osteosarcoma (The same question can be asked with respect to any other bone tumor).

Q. How will you evaluate a case of bone tumor? Discuss the management of giant cell tumor of the bone arising from the upper end of tibia.

Q. What are the types of chondrosarcoma? Discuss the diagnosis and management of chondrosarcoma.

FURTHER READING

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10 Slipped Capital Femoral Epiphysis, Perthes' Disease and Other Osteochondritis

SLIPPED CAPITAL FEMORAL EPIPHYSIS (SCFE)

This condition is known by various names as adolescent coxa vara, epiphyseal coxa vara and epiphyseolisthesis. It refers to an idiopathic slip of the capital femoral epiphysis which occurs commonly during the period of rapid growth, i.e. between 12 and 18 years. The slip may be acute, gradual or acute on a gradual.

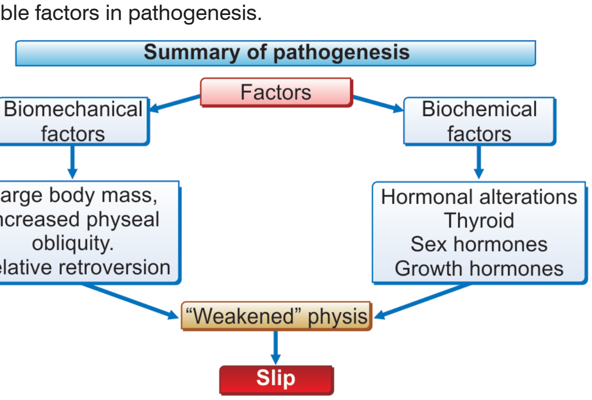
Predisposing Factors (Flow Chart 10.1)

a. Sex—commonly seen in males. M:F ratio 5:1. The left hip is commonly affected than the right hip.
b. Obesity.
c. Weakening and thinning of the physis:
   Exact reason is not known. Thought to be due to malnutrition and in some hormonal imbalance as characterized by relative preponderance of growth hormone. This results in either obese type (due to sex hormone decrease) or Tall type (due to growth hormone increase) of individuals with respective growth abnormality.
d. Altered inclination of the upper femoral physis:
   During adolescence, the inclination of the physis changes from horizontal to oblique.
e. Relative or absolute retroversion of the neck:

Anteversion and retroversion: Angle of inclination of the axis of femoral neck in relation to transcondylar plane is known as version. If the inclination is in anterior direction, it is known as anteversion, antetorsion or

Flow chart 10.1

Possible factors in pathogenesis.



anterior twist. If this angle of inclination is in posterior direction (and the neck points in a posterior direction), it is known as retroversion, retrotorsion or posterior twist. In a newborn, anteversion is as high as 30°. It progressively decreases in childhood and in an adult it is around 10–15° (Fig. 10.1).

Thus, it is postulated that even a normal stress can cause gradual displacement at this weakened physis especially when there is decreased anteversion. In fact, the femoral epiphysis remains in contact with the acetabulum while the neck of the femur which moves anteriorly and superiorly. But, for some reason the slip is described, considering the relationship of the epiphysis (minor fragment) to the femoral neck (major fragment) and is described as 'the epiphysis slips posteriorly and inferiorly' (Fig. 10.2).

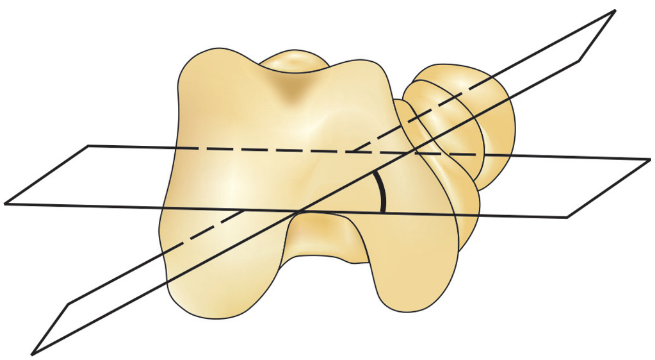


Figure 10.1

Axis of femoral neck and axis of the transcondylar plane by which the ante/retroversion is measured.

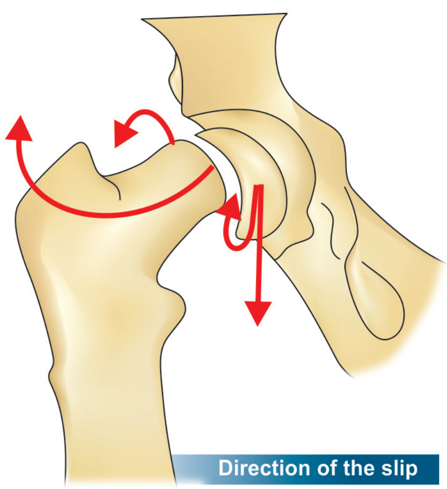


Figure 10.2

Diagrammatic representation of the slip which always occurs in posterior and inferior direction.

Pathology of the Slip (Fig. 10.3)

The slip occurs in the zone of hypertrophic cartilage, or in some (rarely), in the zone of provisional calcification. In these zones, histopathologically, very few normal chondrocytes are seen along with some degenerated and dead chondrocytes. The supportive collagenous matrix is also found to be abnormal.

Whether biochemical/endocrinal factors have caused this change or the mechanical trauma of the slip has resulted in this change, is a debatable issue.

Clinical Features

- An individual in adolescent age with obese or tall built, should increase the suspicion.
- Pain in the hip of gradual onset (at times may present with knee pain).
- Progressive limp.
- Progressive limitation of movement.
- Progressive deformity of adduction and external rotation.
- Presence of 'Axis deviation' sign (Flexion of the hip causes the abduction and external rotation of the limb and the limb falls away from the shoulder).
- Antalgic gait (indicates that the slip is unstable).

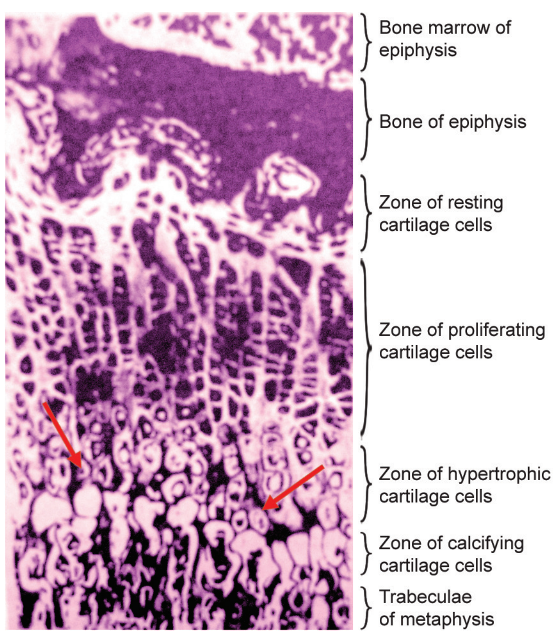


Figure 10.3

Histopathologic section of the physis. Slip always occurs at the zone of hypertrophic and maturing cartilage as shown by red arrow.

Investigations

X-rays of both hips are to be taken as the slip may be bilateral. If unilateral, opposite hip is used for comparison. Standard anteroposterior (AP), lateral and frog leg views of pelvis with both hips are taken (Figs 10.4A to D).

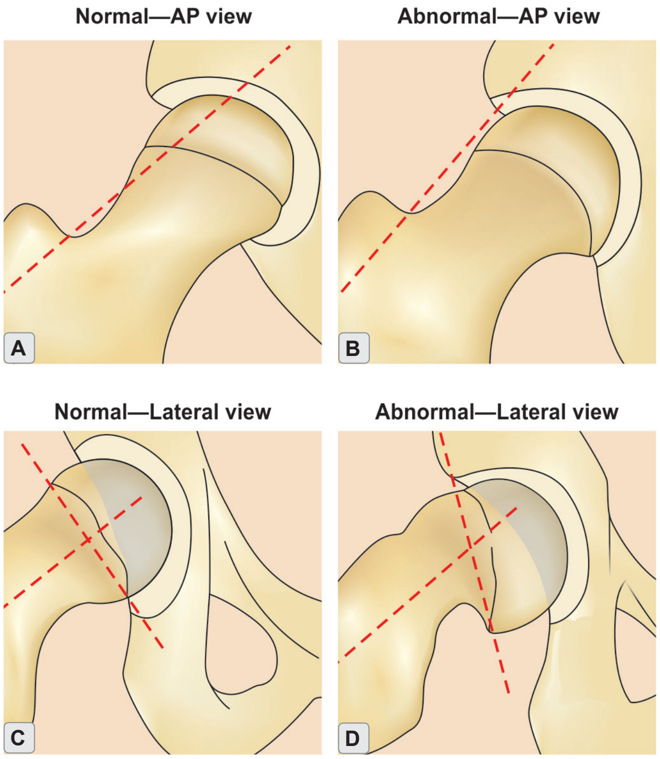
Anteroposterior View

Early sign of slip is indicated by Trethoven's line.

A line drawn along the superior border of the neck of the femur cuts the superior part of the epiphysis in normal hips. In SCFE, it passes over the superior border of the epiphysis (Figs 10.4A and B).

Lateral View

It is more reliable and identifies even the minor degrees of the slip. Two lines are drawn. One bisecting the neck and the other at the base of the epiphysis. In a normal hip these lines are at right angles. An angle less than 90° indicates that the slip has occurred (Figs 10.4C and D).



Figures 10.4A to D

Anteroposterior and lateral views of the hip.
A and C the normal view. In AP view, the Trethoven's line cuts the upper part of the femoral epiphysis and in lateral view, the line at the base of the epiphysis and the line bisecting the neck of the femur are at right angles. B and D the AP and lateral views showing the abnormality. Trethoven's line is passing above the epiphysis in AP view and the bisecting angle is less than 90° in the lateral view. These changes are the early indicators of the slip.

Frog Leg View

A straight line drawn along the center of the femoral neck when continued proximally, passes through the center of the epiphysis in frog leg view. If it passes anteriorly, it indicates SCFE.

Radiological Classification of the Slip

Type I slip—less than 33% displacement.

Type II slip—between 33% and 50% displacement.

Type III slip—greater than 50% displacement (Figs 10.5A to D).

In the long standing cases of SCFE, secondary changes are seen in the head and neck such as avascularity of the epiphysis and malunion of the slipped epiphysis.

Bone scanning, magnetic resonance imaging (MRI), and computed tomography (CT) help in more accurate assessment of the slip and the presence of complications such as AVN, chondrolysis, etc. These investigations, also help in the accurate planning of the surgical procedure.

Treatment

Objectives

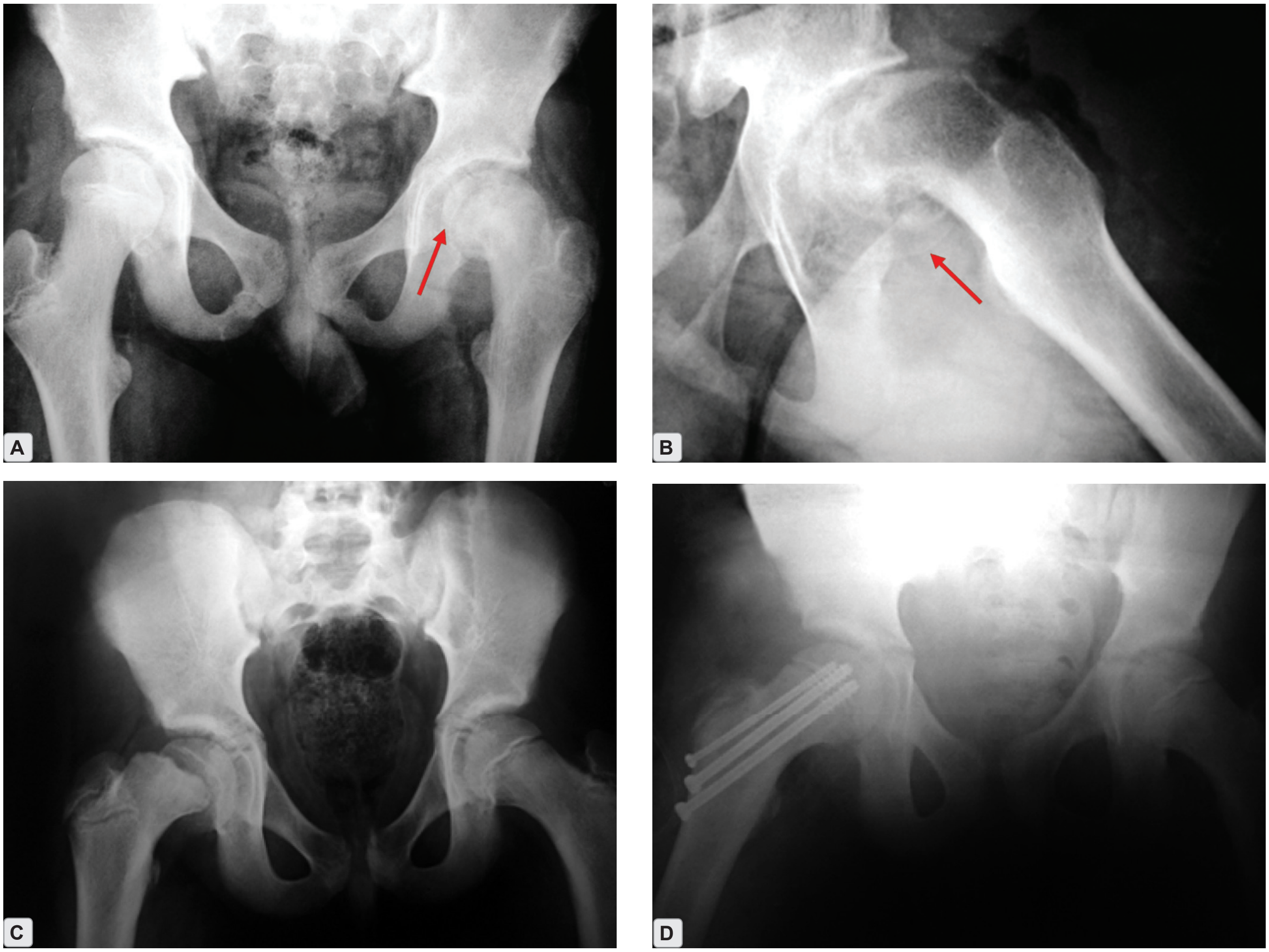
The objectives of treatment are to:

a. Prevent further slip.
b. Achieve premature closure.
c. Reduce the risk of avascular necrosis (AVN) and chondrolysis.

A preliminary traction helps significantly in decreasing the degree of the slip in acute cases.

Surgical Procedures

The procedures are based on the type and the nature of the slip (i.e. the percentage of slip) and whether the slip is stable or unstable. Those slips which present with pain and antalgic gait, are considered unstable and need immediate stabilization; thus, preventing further progression.



Figures 10.5A to D

(A and B) Type III slip, left side. Note the posteroinferior direction of the slip and anterosuperior projection of the neck of the femur; (C) Acute traumatic slip of capital femoral epiphysis, right side; (D) After closed reduction and internal fixation with titanium cannulated cancellous screws.

Cannulated screw fixation and osteotomies: In unstable Types I, II and III, by means of gentle manipulation on a fracture table the slip is reduced and then fixed using a single cannulated screw. The screw fixation helps in premature closure of the physis and prevents further slipping.

In stable type I, the fixation is done *in situ*. It aims at preventing further slip.

In stable type II, choice is between fixation *in situ* and fixation with osteotomy.

In stable type III, osteotomy/epiphysiodesis may be considered.

When further slip occurs it means that the stable slip has become unstable.

Osteotomy: An osteotomy aims at restoring the relationship of the head and the neck of the femur. ***Proximal osteotomies give better correction but are accompanied with the risk of AVN and vice versa.***

Several osteotomies are designed through the apex of the femoral neck, base of the femoral neck and intertrochanteric area, along with or without fixation of the epiphysis to the neck of the femur (Fig. 10.6).

Bone peg epiphysiodesis:

Aim*:* The procedure aims at achieving a premature closure of the epiphysis.

Procedure: In this procedure, corticocancellous graft obtained from the iliac crest, is sandwiched and driven

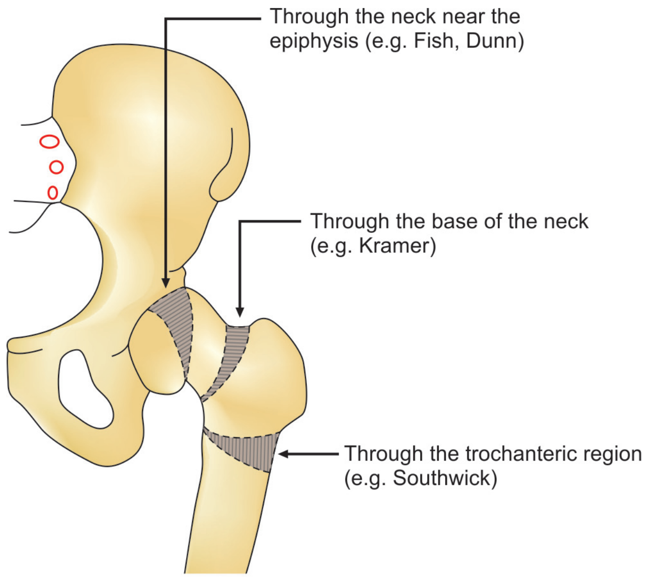


Figure 10.6

Diagrammatic representation of the sites of different osteotomies that are done for SCFE.

into a tunnel created in the epiphysis through a window created in the neck of the femur. Lower incidence of AVN and absence of implant related complications (as no implants are used) are distinct advantages of this procedure (Figs 10.7A and B).

Prognosis: Stable slips have better prognosis following treatment.

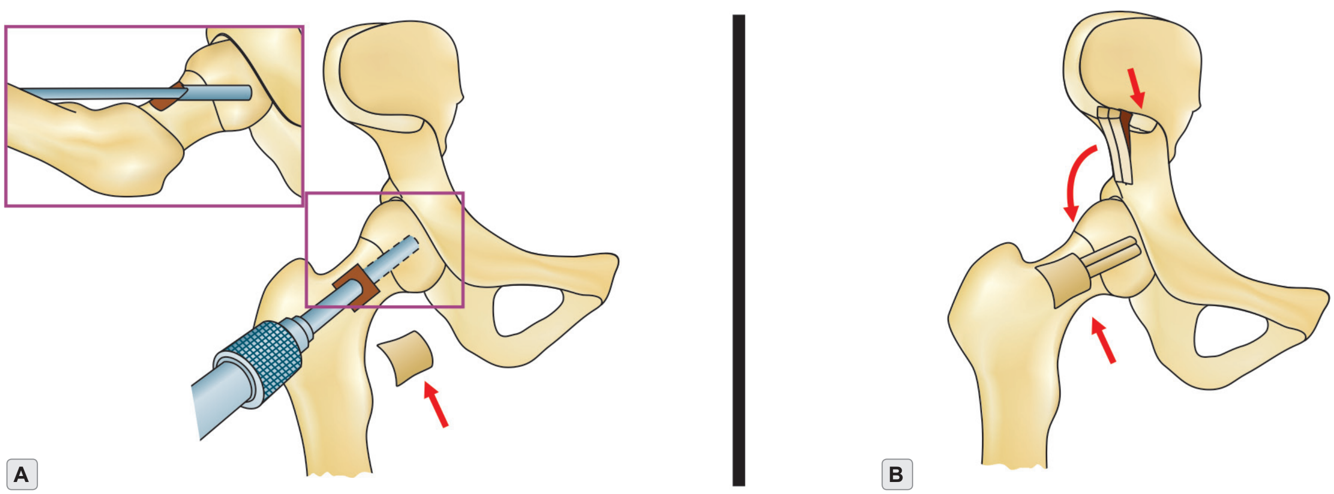
- Unstable slips have a higher risk of poor prognosis following treatment. This is the result of damage to epiphysial blood supply at the time of acute slip and not because of the surgical procedure.
- Similarly, early presentation and minor degrees of slip have better prognosis than late presentation and more severe degrees of slip.

Prophylactic pinning of the opposite hip: Prophylactic pinning is indicated based on 'Oxford bone score'. This score is based on the stages of development of ilium, triradiate cartilage, head of the femur, greater trochanter and lesser trochanter. A score of around 16 implies 85% chance of slip in contralateral epiphysis.

Correction of residual deformity after closure of the physis is not recommended because there is not enough proof that it helps in preventing the late complications.

Summary of management of different stages of SCFE is shown in Flow chart 10.2.

Complications: Chondrolysis and avascular necrosis are the late complications of SCFE. When the pain is severe and functional limitation that occurs is troublesome either a total joint arthroplasty or an arthrodesis are the procedures of choice.

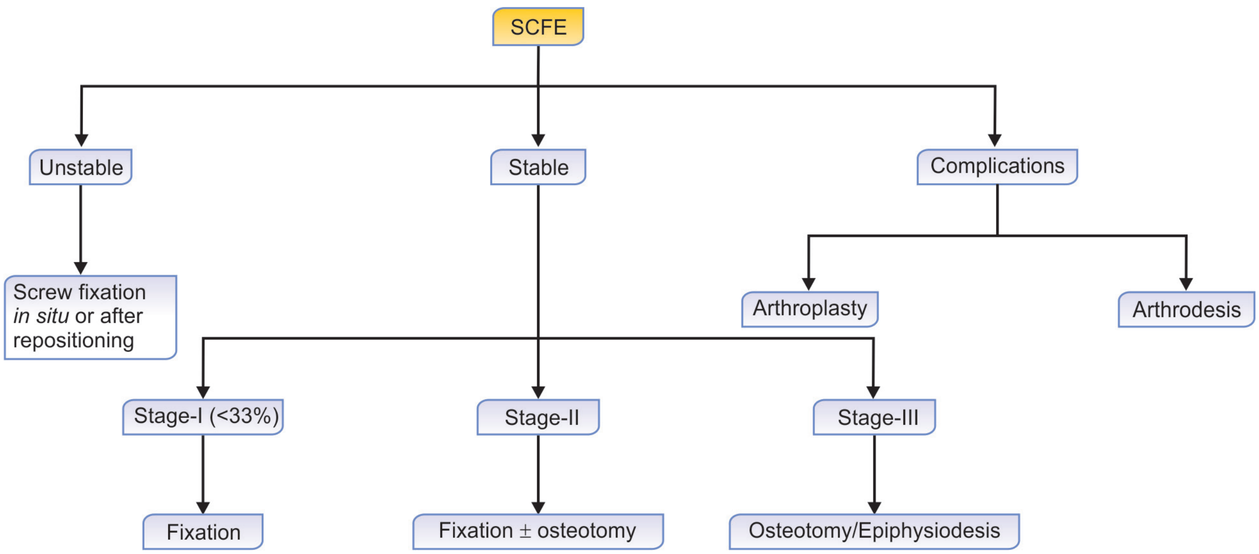


Figures 10.7A and B

(A) Preparation of the neck and epiphysis of the femur by lifting an osteomized rectangular block of bone and (B) insertion of sandwiched iliac crest graft for achieving epiphysiodesis followed by placing the bone block back in its place.

Flow chart 10.2

Managment of slipped capital femoral epiphysis.



Revision Questions

- Q. What are the other names for slipped capital femoral epiphysis?
- Q. How will you classify the SCFE?
- Q. Discuss the etiopathogenesis of SCFE.
- Q. Draw Trethoven's line. What is its importance?
- Q. Discuss the management of SCFE.
- Q. What are the complications of SCFE? How will you manage them?

Essay Question

- Q. Discuss the etiopathogenesis of slipped capital femoral epiphysis. How will you investigate, diagnose and treat a case of slipped capital femoral epiphysis. Enumerate its complications.

LEGG-CALVE-PERTHES' DISEASE

Arthur Legg in 1909 described the disease in the annual meeting of American Orthopedic Association at Hartford, Connecticut, USA. By the time he published his work in 1910, Jacques Calve of France and George Clemens Perthes of Germany had also published their cases. Thus the eponym 'Legg-Calve-Perthes' originated.

Though, Henning Waldenström had first described the radiological changes of this disease, he thought those were a type of benign tuberculosis. The disease is also known by different names such as osteochondritis deformans juvenilis, pseudocoxalgia and coxa plana.

It is a disease occurring in children between the age of 5–15 years characterized by varying degrees of idiopathic avascular necrosis of the upper femoral epiphysis followed by spontaneous revascularization over a period of time, i.e 1½–3 years. During this period, the head is exposed to a risk of deformation. Hence, needs protection from deforming forces (see Figs 10.15A and B).

Predisposing Factors

Following are the factors observed.

1. *Genetic:* The disease shows a male predominance with a M:F ratio 5:1. Also, the incidence is higher among later siblings. The Asians, Eskimos and Whites have a higher incidence when compared to Australian aborigines, Polynesians, American Indians and African Americans.
2. *Abnormal growth and development:* The bone age in patients with Perthes' disease is found to be lower than their chronologic age by 1–3 years.
3. *Environmental factors:* Higher percentage of involvement is seen among the lower socioeconomic group.
4. *Trauma.*
5. *Transient synovitis.*
6. *Passive smoking.*
7. *Abnormal venous drainage.*
8. *Arterial block and infarction*.
9. *Increased viscosity of the blood*.

Pathology

The pathology of idiopathic avascular necrosis followed by spontaneous revascularization passes through 4 stages:

1st stage—Stage of synovitis: In this stage, the synovium is swollen, edematous and thickened. There is effusion into the joint. But, no inflammatory cells are found in the fluid.

2nd stage—Stage of avascular necrosis: In this stage, the trabeculae are crushed and compressed into a compact mass. Debris filled spaces as well as vacant lacunae, are seen. Metaphyseal hypervascularity is evident. Articular cartilage remains intact and shape of the head is maintained.

3rd stage—Stage of (fragmentation) revascularization: In this stage, highly vascular connective tissue invades the area and the necrotic tissue is replaced by osteogenic tissue. Occurs over a period of 1½–3 years.

4th stage—Residual/healed stage: All the necrotic bone is replaced by mature bone.

The metaphysis also shows certain changes such as lytic areas with sclerotic margins, abundant fatty marrow, disarrayed ossification process, etc.

Radiological Features

The pathological changes that occur, produce certain characteristic radiological features.

Thus, the disease has been staged by Elizabethtown staging into 4 stages and grouped by Catterall into 4 groups.

Based on Elizabethtown Staging (Figs 10.8A to D)

Stage I (Initial/Avascular necrosis): Shows condensation, compression and increased density of the epiphysis with widening of the medial joint space (Waldenstrom's sign).

Stage II (Fragmentation): Further shrinkage and fragmentation of the epiphysis is seen. It indicates an intermediate reparative phase.

Stage III (Regeneration): Shows more pronounced fragmentation along with new bone formation. It indicates healing phase of the disease.

Stage IV (Healed): Complete re-ossification. No fragmentation or increased density observed.

Based on Catterall Grouping (Figs 10.9A to D)

The disease is grouped into 4 groups depending on:

- Extent of epiphyseal involvement.
- Presence of sequestrum.
- Presence of collapse.
- Metaphyseal abnormality.

It is summarized in Table 10.1.

Classifications Useful to Assess Prognosis

Based on Herring's lateral pillar classification: In this, the epiphysis is divided into three pillars; medial, central and lateral respectively (Figs 10.10A to C).

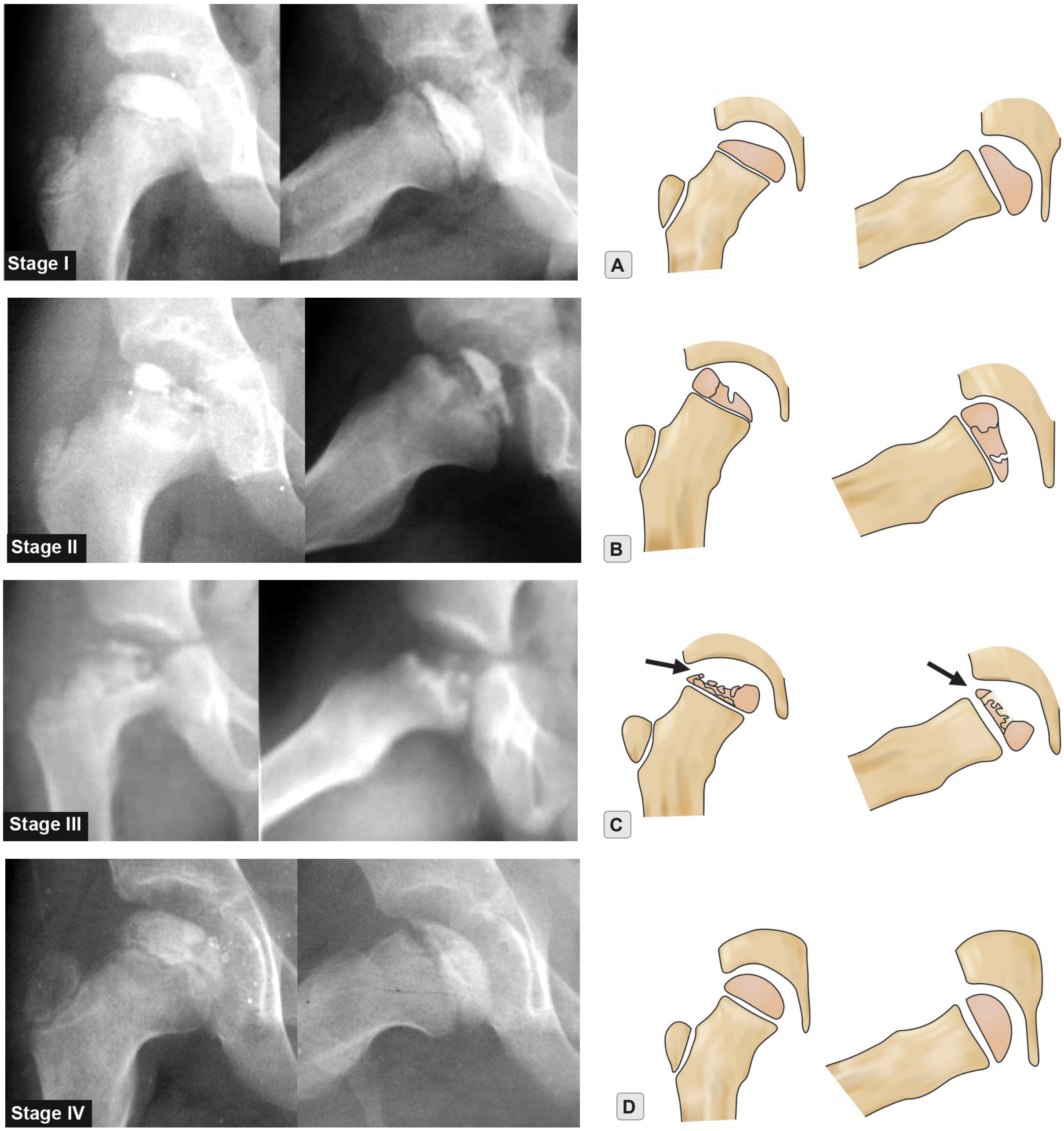
The classification is based on the changes that occur in the lateral portion of the femoral head/epiphysis (lateral pillar) when early fragmentation begins. When the lateral pillar is intact, it acts as a weight-bearing support to protect the central avascular fragment. When lateral pillar is involved and collapsed the weight-bearing stresses damage the epiphysis rapidly.

Salter and Thomson classification: This is based on the presence, location and extent of subchondral fracture. Four groups are identified. Prognosis is directly proportional to the chronological order of the classification.

Table 10.1

The portion and nature of involvement of the epiphysis and metaphysis in different groups based on Catterall's classification.

	Group I	*Group II*	*Group III*	*Group IV*
Epiphyseal involvement	Anterior and lateral part	More anterior part	Entire epiphysis except a small posterior part	Entire epiphysis
Sequestrum	No	Yes	Yes	Yes
Collapse	No	Yes	Yes	Yes
Metaphyseal abnormality	No	Localized	Diffuse	Diffuse



Figures 10.8A to D

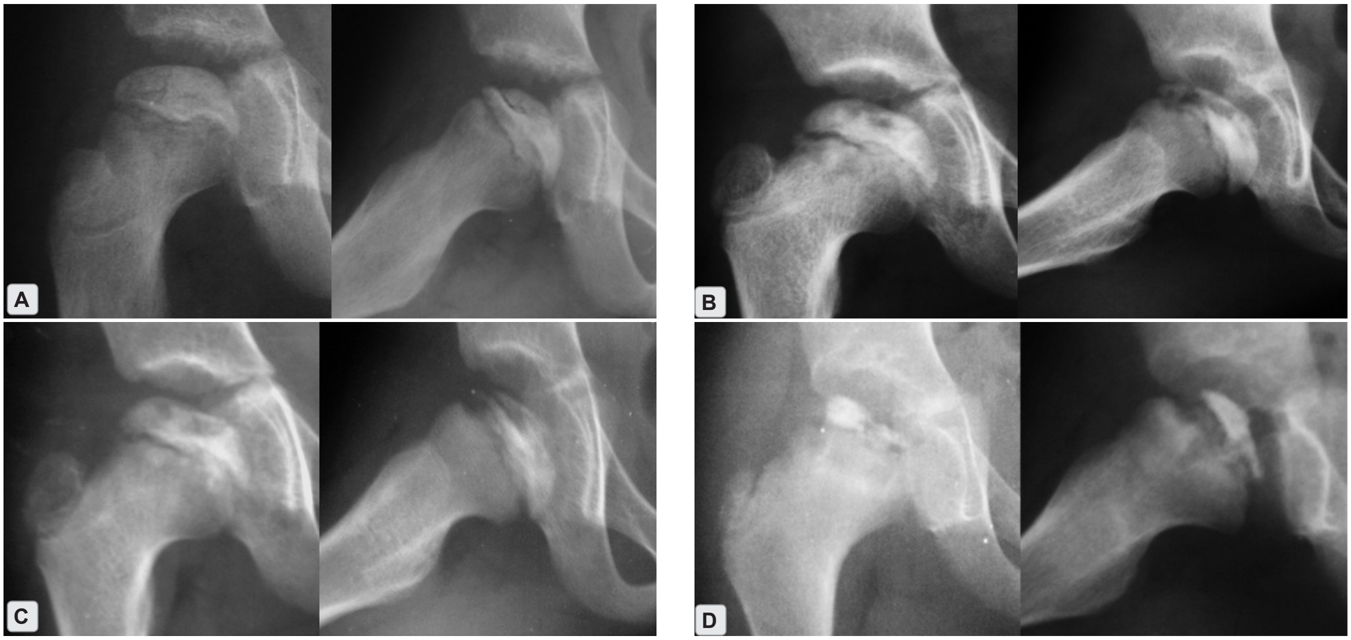
Radiographs of different clinical stages of Perthes' disease as per Elizabethtown staging and diagrammatic representation of Elizabethtown staging.

Clinical Features

Onset is insidious. Presenting features are as follows:

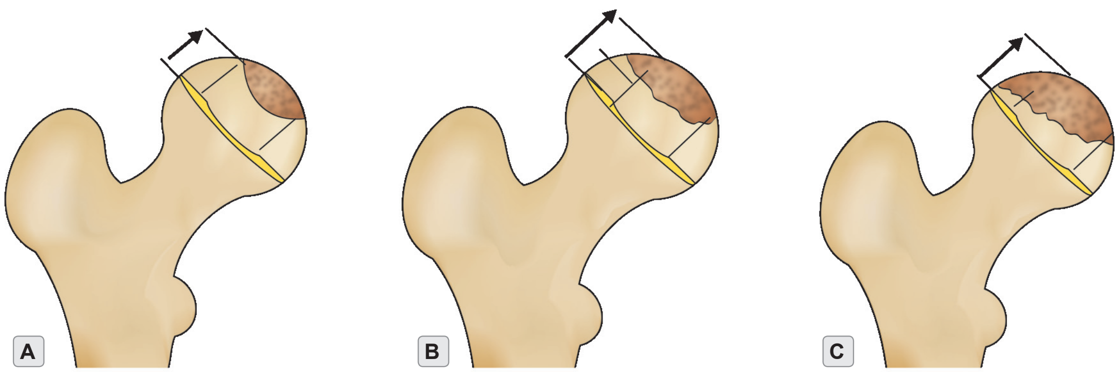
1. Limp with or without dull pain.
2. Pain in the hip region. Medially in the groin, laterally, in the region of the greater trochanter and anteriorly, in the inguinal region. At times, radiates to the knee.
3. Pain is exaggerated by activity, relieved by rest.
4. Classical limitation of abduction and internal rotation.
5. Trendelenburg gait may be present.

Note: *'Head within the Head' is a radiological sign seen in some cases of Catterall Group-III, Perthes' disease. This radiological image appears because of superimposition of image of small, posterior portion of the head which is vascular, on majority of the head which is avascular.*



Figures 10.9A to D

Anteroposterior and lateral radiographs showing different groups according to extent of head involvement based on classification by Catterall. A-Group I; B-Group II; C-Group III; D-Group IV. Note the 'head within the head' sign in Group III.



Figures 10.10A to C

Showing the fragmentation based on lateral pillar concept of Herring: (A) Only central pillar fregmentation. The lateral pillar protects the fragmented part; (B) Lateral pillar fragmentation < 50% of the height of the epiphysis; and (C) Lateral pillar fragmentation > 50% of the height of the epiphysis.

Head at Risk Signs

Clinical "Head at Risk" Signs

1. Older child
2. Heavy child
3. Progressive loss of movement
4. Adduction contracture
5. Flexion with adduction deformity.

Radiological "Head at Risk" Signs (Fig. 10.11)

1. Gage sign (1933)
   It is a 'V' shaped radiolucent defect on the lateral side of the epiphysis.
2. Calcification lateral to the epiphysis.
   Occurs due to thickened and extruded epiphysis. Indicates epiphyseal enlargement.
3. Lateral subluxation.
4. Horizontal growth plate.
5. Diffuse metaphyseal lesion.

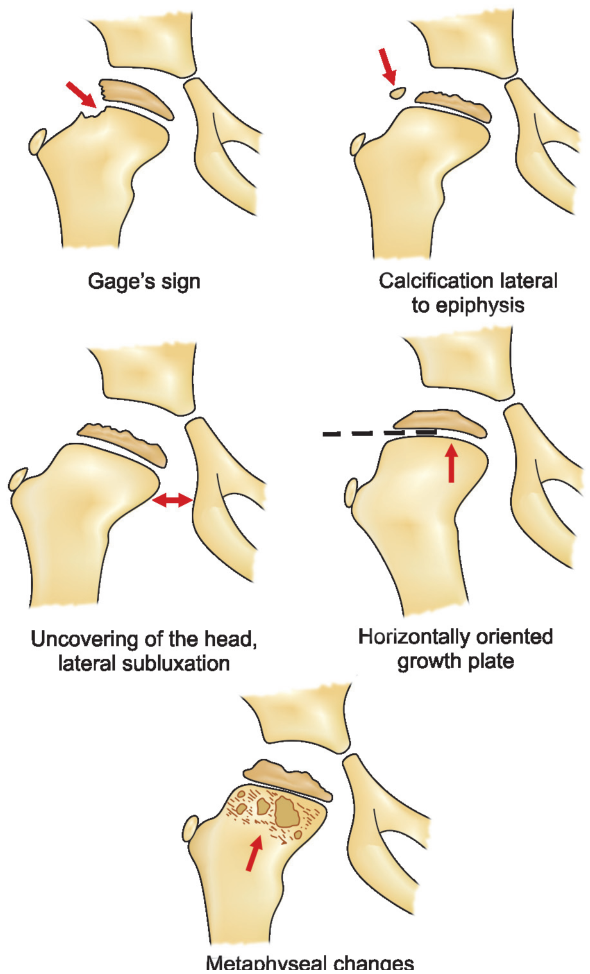


Figure 10.11

Diagrammatic representation of 'head at risk' signs.

Among these the only pathology that is amenable for treatment is the lateral subluxation (Fig. 10.11). Hence this may be considered as the only 'head at risk' sign. All other signs though considered as signs which can contribute to bad prognosis are in fact not amenable for correction.

Investigations

1. X-ray: Shows avascular changes as per stage of the disease.
2. Bone scan: Can diagnose the avascularity much before the radiological changes develop. Also useful in differentiating septic from aseptic inflammation.
3. MRI: Delineates the changes in the cartilaginous epiphysis and the joint much better than X-ray. Also useful to assess vascularity of the epiphysis.
4. Arthrography: Useful in the assessment of joint congruity and helps in planning the containment procedure.

Treatment

Aim: Is to prevent the damage to the vulnerable, avascular epiphysis by protecting it till it gets fully vascularized.

Such an environment is created either nonoperatively or operatively, whichever is appropriate for the manner of presentation. The procedure is known as 'containment'. The procedure/method contains the epiphysis within the confines of the acetabulum and takes off the abnormal stresses to which it is otherwise subjected to, till complete revascularization takes place.

Effective treatment, minimizes the risk of complications of growth disturbance and secondary degenerative arthritis as a sequel of avascular necrosis.

Factors to be Considered in Planning the Treatment

1. Age of the patient:

   <5 yr—consider nonsurgical containment only if subluxation is observed.

   5–7 yr—consider nonsurgical/surgical containment only if subluxation is observed.

   7–12 yr—consider nonsurgical/surgical containment before lateral subluxation occurs (when other head at risk signs are present). This is the age where the head is prone to subluxation. Hence, treatment should be more aggressive to prevent subluxation.

   >12 yr—do not consider containment.
2. Range of hip movement:

   If hip movements are near normal, consider containment. If hip movements are restricted, do not consider containment.
3. Stage of the disease:

   In Elizabethtown stages I and II, consider containment. In stages III and IV, do not consider containment.
4. Extent of epiphyseal involvement:

   Consider containment if half or more than half of the epiphysis is involved (Catterall group II, III and IV).

   If less than half of the epiphysis is involved, do not consider containment.

To summarize, surgical containment is considered in a vulnerable epiphysis, in a child of 7–12 years of age, with no restriction of hip movements; the stage of the disease according to Elizabethtown staging is stage I or II and the extent of involvement as per Catterall clasification is group II, III or IV (Refer: Further reading reference 41–45 for more information).

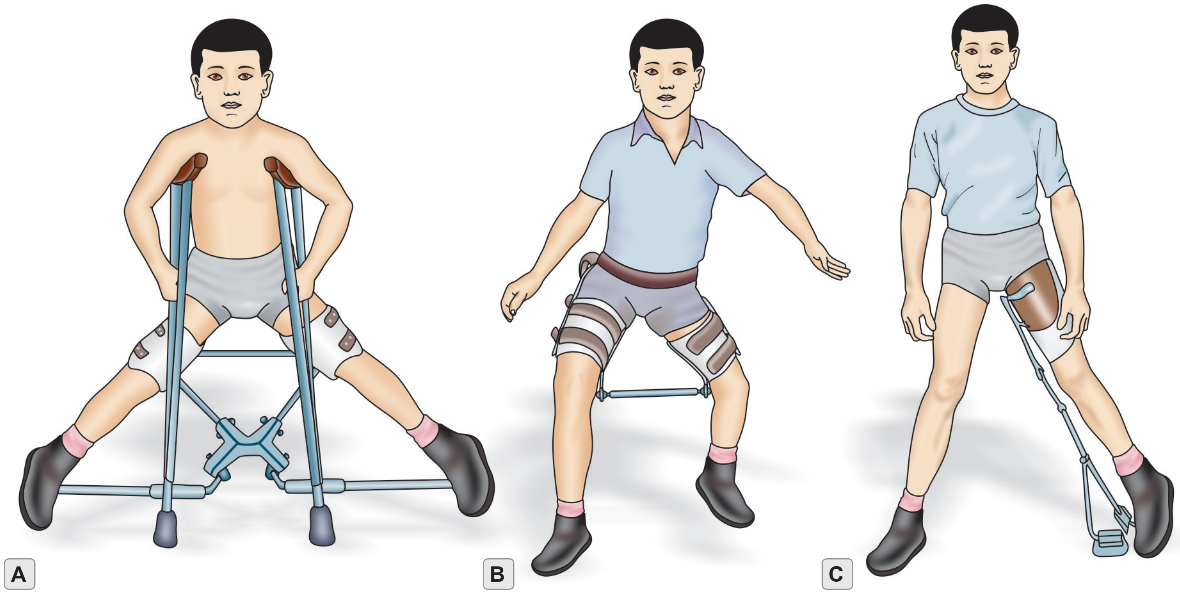
Nonsurgical Containment Methods

A. Nonambulatory
   - Bedrest and traction in abduction
   - Broom stick plaster
   - Hip spica cast

B. Ambulatory (Figs 10.12A to C)
   - Newington abduction frame orthosis
   - Toronto brace
   - Scottish-Rite orthosis
   - Tachdjian brace (Trilateral socket hip containment orthosis)

Surgical Methods of Containment

A. Femoral varus derotation osteotomy (Fig. 10.13)
B. Salter's innominate osteotomy (Fig. 10.14)



Figures 10.12A to C

Nonsurgical method of containment with the help of braces which aim at keeping the hip in about 40–45° of abduction. Some of the popular designs are: (A) Toronto brace; (B) Atlanta Scottish Rite Children's Hospital brace; and (C) Tachdjian abduction brace.

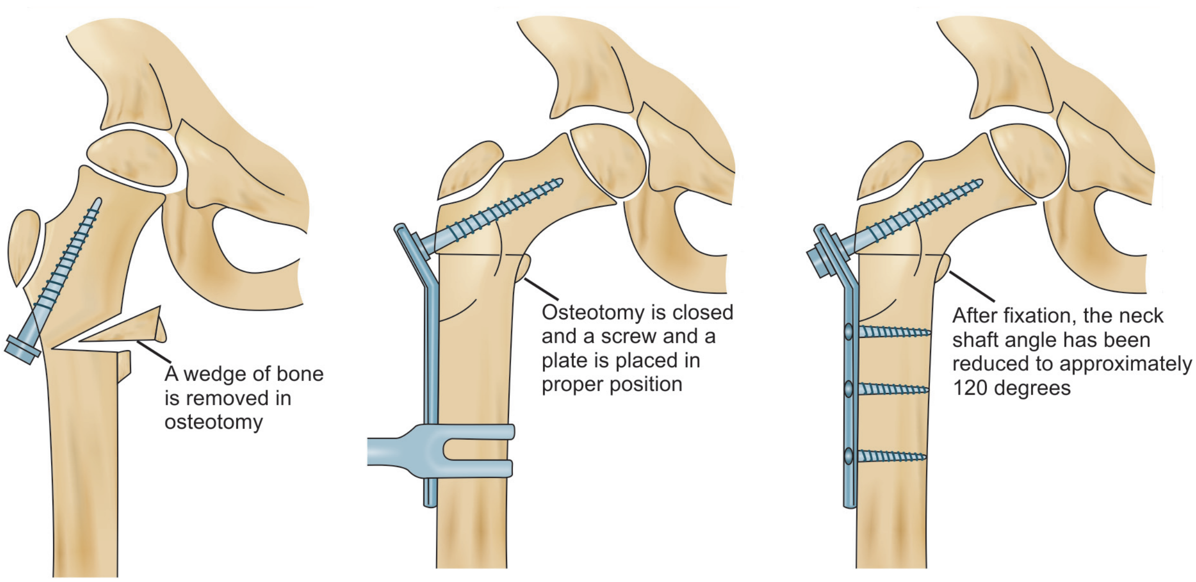


Figure 10.13

Diagrammatic representation of medial closed wedge osteotomy for containment (The containment may also be achieved by lateral open wedge osteotomy).

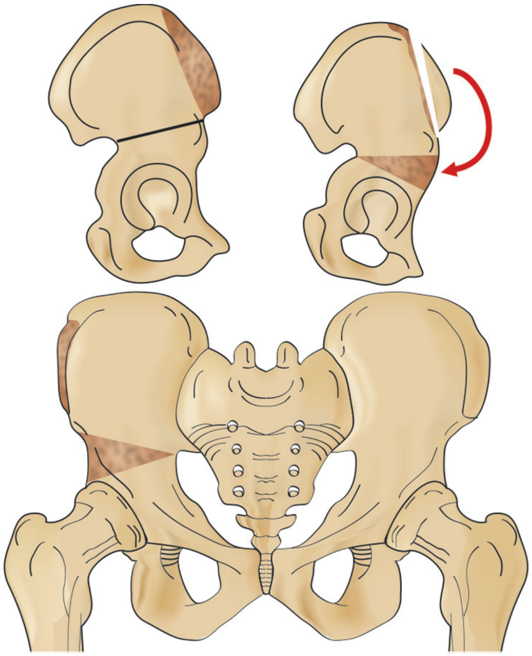
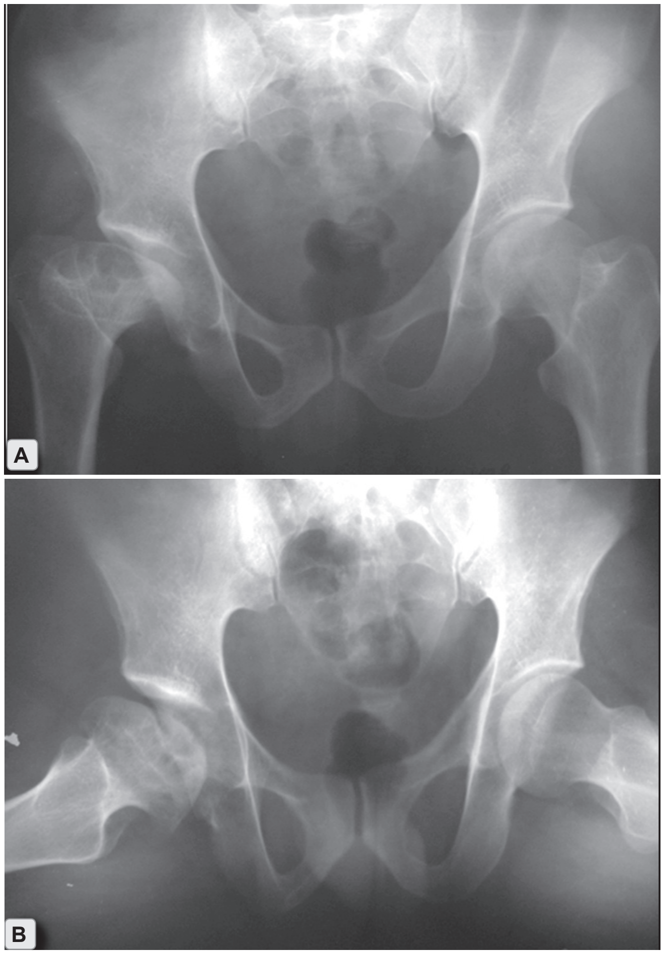


Figure 10.14

Salter's innominate osteotomy procedure for containment of the epiphysis of the femur. Note that the osteotomy is done just above the roof of the acetabulum and opened. A triangular wedge of bone from the iliac crest is taken and inserted at the opened site and fixed.

Prognostic Factors

- Age of onset.
  Early onset has better prognosis than late onset.
- Female gender.
  Said to have poor prognosis when compared to male.
- Remodeling potential.
  Varies from person to person. Greater the remodeling, better is the prognosis.
- Protracted course.
  If the course of the disease is protracted, the prognosis is poor.
- Growth disturbance (Figs 10.15A and B).
  Premature closure of the physis leads to growth disturbance and may affect the outcome.
- Stage at which the patient presents for treatment.
  If the patient presents for treatment late, the epiphysis would already have been deformed. This results in poor prognosis.



Figures 10.15A and B

Radiographs AP and Frog leg view of pelvis with both hips showing late sequel of Perthes' disease. Note the irregularity and mushrooming of the head of the femur and the adaptive changes in the acetabulum in relation to the shape and size of the head. Both are incongruous when compared to the normal head but between them have maintained the congruity (incongruous congruity). Patient was walking with a pain free limp with shortening, i.e with a short limb gait. He is at risk for development of secondary degenerative arthritis at a later period.

Revision Questions

Q. Define Legg-Calve-Perthes' disease.
Q. Discuss the stages of Perthes' disease.
Q. Discuss the etiopathogenesis of Perthes' disease.
Q. What are the head at risk signs? Enumerate them.
Q. What is Waldenström's sign?
Q. Head within the head sign.
Q. Write briefly on classification of Perthes' disease.

Essay Question

Q. Discuss the etiopathogenesis diagnosis and management of a case of Perthes' disease. Enumerate the complications.

IDIOPATHIC AVASCULAR NECROSIS/OSTEOCHONDRITIS

The exact etiology of these diseases is not known. Hence, they are grouped under idiopathic avascular necrosis. The various factors observed in different areas definitely indicate the role of mechanical factors in the origin and progress of the disease. Abnormal blood supply, increased viscosity of the blood, infarction and increased intraosseous pressure are the other factors identified. Thus, it is wise to conclude that the origin and the progress of these diseases is multifactorial and so are the modalities of treatment. It varies from simple stress relieving measures such as restricted weight-bearing, abstaining from strenuous activities, protective splints, etc. to surgical methods. Few of the common diseases are discussed here.

Bones involved commonly in idiopathic avascular necrosis/osteochondritis

Bone involved	*Name of the disease*
Scaphoid	Preiser's disease
Capitulum	Panner's disease
Lunate	Kienbock's disease
2nd metatarsal head	Freiberg's disease (infarction)
Navicular	Kohler's disease
Calcaneal apophysis	Sever's disease
Tibial tuberosity	Osgood Schlatter's disease
Upper femoral epiphysis	Legg-Calve-Perthes' disease
Ring epiphysis of the vertebra	Scheurmann's disease
Central epiphysis of the vertebra	Calve's disease

Kienbock's Disease

It is avascular necrosis of the carpal lunate. It was first described by Robert Kienbock in the year 1910. He called it 'lunatomalacia'.

Etiology

Exact etiology is yet to be ascertained. It is considered as multifactorial. Certain predisposing factors have been identified. These factors perhaps predispose lunate to excessive/abnormal stresses, resulting in damage to the blood supply and avascular necrosis.

a. Ulnar variant (Hulten, 1928)

   Ulnar-neutral—the distal radius and ulna are at the same level.

   Ulnar-plus—the distal ulna is farther than the radius.

   Ulnar-minus—the distal ulna is shorter than radius.

   In Kienbock's disease ulnar-minus variant has been commonly observed.

b. Shape of the lunate (Antuna Zepico, 1966)

   Type I—lunate with a proximal apex or crest.

   Type II—lunate is more squarish.

   Type III—lunate is more rectangular.

   In Kienbock's disease with ulnar-minus variant, Type I lunate is observed. Whereas:

   Type II and Type III lunates are seen in ulnar-neutral and ulnar-plus variants.

c. Slope of the distal articular surface of the radius (Mirabello, Werner, Palmer)

   In Kienbock's disease, the normal inclination of the articular surface of the lower radius is found to be decreased.

d. Primary circulatory disturbances and increased intraosseous pressure, are also considered as predisposing factors.

Investigations

X-ray: Shows increased density of the lunate suggesting avascularity. Fractures and fissuring, carpal collapse and degenerative changes are seen as the disease progresses.

MRI: Delineates the abnormality more clearly.

Treatment

Treatment depends upon the age of the patient, the state of the lunate and the carpus.

Early: In early disease, if mechanical variants are present they are corrected by osteotomy and radial shortening procedures.

Late: In advanced cases, where degenerative changes are present, scaphotrapezio-triquetral/scaphocapitate arthrodesis or wrist arthrodeisis is indicated.

(Nonoperative treatment in the form of prolonged immobilization is known to cause progression of the disease. Hence its use is questionable).

Kohler's Disease

It is avascular necrosis (osteochondritis) of the tarsal navicular. First described by Kohler in 1908, the disease is self-limiting and invariably resolves over a period of time. The disease is more common in boys than girls. It is common between 2 and 9 years (Figs 10.16A and B).

Etiology

a. Repetitive mechanical stress is thought to produce microfractures and avascularity.
b. Abnormal vascularity of the navicular.

Investigations

X-ray for diagnosis and MRI for confirmation of avascularity.

Figures 10.16A and B

(A) Radiograph of normal foot showing a normal navicular bone placed for comparison; (B) Kohler's disease. Note the increased density and fragmentation and the small size of the navicular bone.

Treatment

The disease should be treated by restricted weight-bearing and protection in cast and splint. Healing invariably occurs over a period of time.

Revision Questions

Q. Write briefly on osteochondritis. Enumerate different osteochondritis.
Q. How will you manage a case of osteochondritis?

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Perthes' Disease

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11 Intervertebral Disc Prolapse

- *Structure and function of the disc*
- *Terminologies and types*
- *Investigations*
- *Disc replacement, its current status*
- *Pathomechanics of disc prolapse*
- *Clinical presentation: symptoms, signs and diagnosis*
- *Treatment*

INTRODUCTION

Intervertebral discs constitute approximately 1/4th of the vertebral column. They are designed to absorb shocks when the spinal column is subjected to mechanical loading during the activities of daily living. A disc has a central, gel-like nucleus pulposus made up of collagen, proteoglycans and water. It is placed somewhat like a cushion between the cartilaginous end-plates of the adjacent vertebra. The nucleus pulposus is surrounded by annulus fibrosus which is made up of concentric sheets of collagen fibers connected to vertebral end-plates. These concentric sheets are held firmly by radial fibers which are placed at intervals (Figs 11.1A to C).

Pathomechanics of Intervertebral Disc Prolapse

When the nucleus pulposus invaginates through the annulus fibrosus the condition is known as prolapsed intervertebral disc. In milder forms this results only in a protrusion. But, in severe forms it extrudes into the spinal canal and later gets sequestrated. ***For a prolapse to occur there should be a breach in the annulus fibrosus.***

A breach in the annulus can develop as a result of:

a. *Severe and sudden trauma, e.g. lifting very heavy loads.* Bending and torsional forces that ensue, cause a sudden increase in pressure within the disc and rupture the annulus (Figs 11.2A and B).
b. *Trivial trauma like coughing and sneezing: With advancing age repetitive stresses cause degenerative changes both in annulus fibrosus and nucleus pulposus. When such a disc which is already weakened, is subjected to trivial trauma, it prolapses.*

The different stages of a disc prolapse are as follows (Figs 11.3A to C):

$$\left.\begin{array}{ll}\text{a.} & \text{Protrusion}\\ \text{b.} & \text{Extrusion}\\ \text{c.} & \text{Sequestration}\end{array}\right\}\ \text{Prolapse}$$

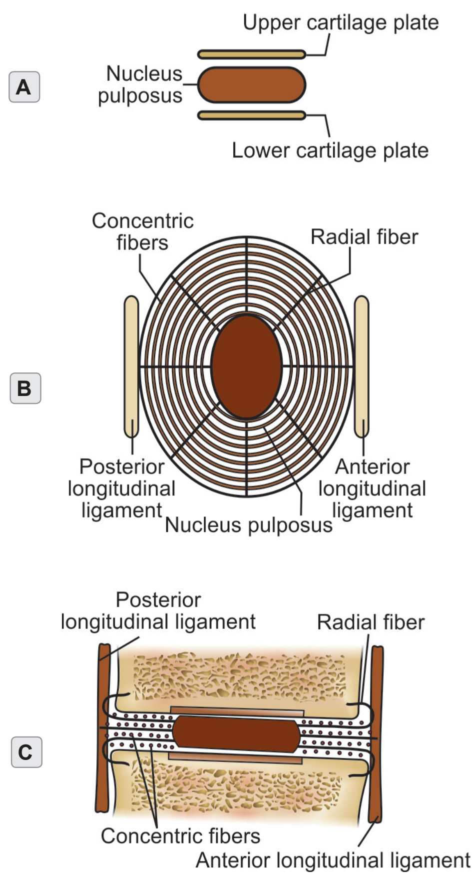
In the lumbar region, L4–L5 is the most common site for disc prolapse followed by L5–S1. These are sites which are prone for maximum concentration of stresses during daily activities involving spinal loading.

In the cervical spine, most of the disc prolapses are degenerative. Purely traumatic disc prolapse without spinal column injury is rare. C5, C6 and C6, C7 are the common sites.

A prolapsed disc occupies various positions in relation to the nerve root and compresses the root.

a. It may remain central.
b. It may remain medial to the root.
c. It may remain lateral to the root.
d. It may remain intraforaminal (rare).

Depending on the position the symptoms vary. Sometimes, disc prolapse can occur at multiple levels.



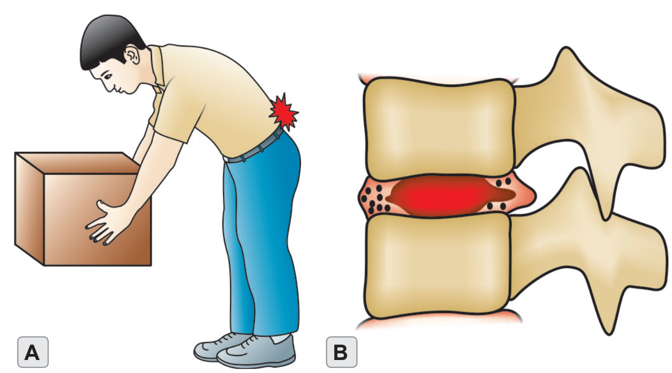
Figures 11.1A to C

Diagrammatic representation of the basic structure of an intervertebral disc.

DIAGNOSIS

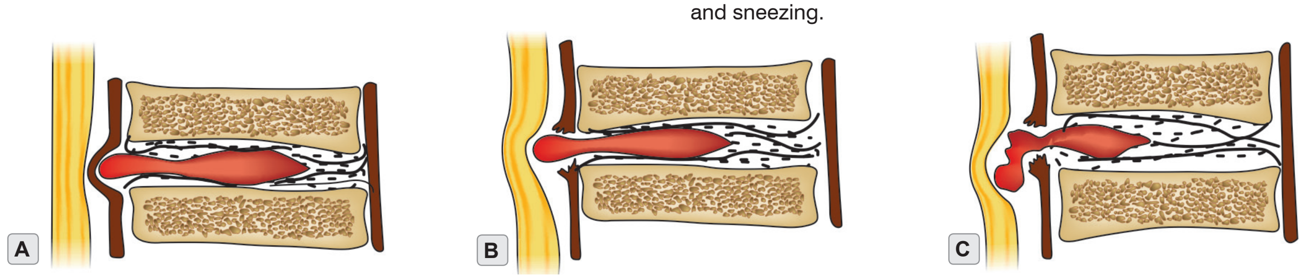
Clinical diagnosis of intervertebral disc prolapse is based on the symptoms and deficits caused by the compression of the nerve roots.

1. **When there is no neurological involvement:** It manifests with acute onset of severe low backache and paraspinal spasm. Movements of the spine also may be restricted and associated with pain. Patient may have difficulty in walking too.
2. **When there is neurological involvement:** Along with pain and limitation of movement, there is neurological deficit. Derivation is almost mathematical and corresponds to the particular



Figures 11.2A and B

Diagrammatic representation of the pathomechanics of disc prolapse: (A) Disc subjected to sudden trauma of lifting heavy load; (B) Degenerative disc which is more prone for prolapse when subjected to stresses of heavy loading as well as milder stresses of coughing and sneezing.



Figures 11.3A to C

Stages of disc herniation, namely protrusion, extrusion and sequestration. Note that the compression on the spinal cord is more with extrusion and sequestration. So also is the indication for surgery.

nerve root affected. Hence, a thorough knowledge of the dermatomes and root values of the nerve supply to the muscles is necessary not only to make a clinical diagnosis, but also to ascertain the level of the lesion.

In cauda equina lesions due to central herniation of the disc, presentation is classic. It presents with leg pain, perianal anesthesia, weakness of the anal sphincter and urinary retention.

Tables 11.1 and 11.2 summarize methodically, the clinical manifestations of a disc prolapse at various levels. Figure 11.4 is a pictorial representation of different examination findings of lumbar disc prolapse at different levels.

Presence of a List or Tilt in the Spinal Column

When the extruded disc lies medial to the nerve root and compresses the nerve root from its medial side

Table 11.1

Clinical features of cervical disc lesions at different levels.

	C-4, C-5 disc with C-5 root compression	*C-5, C-6 disc with C-6 root compression*	*C-6, C-7 disc with C-7 root compression*	*C-7, T-1 disc with C-8 root compression*	*T-1, T-2 disc with T-1 root compression*
Sensory changes (Deficit)	Lateral aspect of upper arm and elbow	Lateral aspect of the forearm thumb and the index finger	Usually middle finger but variable due to crossover	Ring and little finger along with ulnar border of the hand	Medial aspect of the elbow
Motor changes (Weakness)	Deltoid and biceps brachii	Biceps brachii, extensor carpi radialis longus and brevis	Triceps, flexor carpi radialis, flexor digitorum superficialis and profundus	Interossei and the finger flexors, flexor carpi ulnaris	Interossei
Reflexes	Absence of or sluggish biceps reflex	Absence or sluggish biceps and brachioradialis reflex	Absence or sluggish triceps reflex	No change	No change

Table 11.2

Clinical features of lumbar disc lesions at different levels.

	Ruptured disc between L-3 and L-4 with L4 root compression (L-4 component of femoral and sciatic nerve)	*Ruptured disc between L-4 and L-5 with L-5 root compression (L-5 component of the sciatic nerve)*	*Ruptured disc between L-5 and S-1 with S-1 root compression (S-1 component of the sciatic nerve)*
Pain	SI joint* and hip region. Posterolateral aspect of the thigh. Anterior aspect of the leg	SI joint and hip region. Posterolateral aspect of the thigh and leg	SI joint and hip region. Posterolateral aspect of the thigh, leg, and heel
Numbness	Anteromedial aspect of the leg	Lateral aspect of the leg or dorsum of the foot extending over great toe	Lateral aspect of the leg, foot and lateral three toes
Weakness	Extension of the knee	Dorsiflexion of great toe and occasionally foot	Not common. Occasionally plantar flexion of foot and great toe
Sensory changes (Deficit)	Posterolateral aspect of the thigh, anterior aspect of knee and medial aspect of the leg	Anterolateral aspect of the leg, dorsum of the foot and great toe	Lateral malleolus, lateral aspect of the foot and heel and dorsum of lateral one and a half toes including web
Motor changes (Weakness)	Quadriceps and hip adductors	Extensor hallucis longus, extensor digitorum longus and brevis; gluteus medius at the hip	Gastrocnemius and soleus, peroneus longus and brevis; gluteus maximus at the hip
Reflexes	Sluggish or absent knee jerk	No change both in knee and ankle jerk (or sometimes sluggish reflex). Never absent. Tibialis posterior reflex may be sluggish/absent	Sluggish or absent ankle jerk

*SI Joint—Sacro-iliac joint

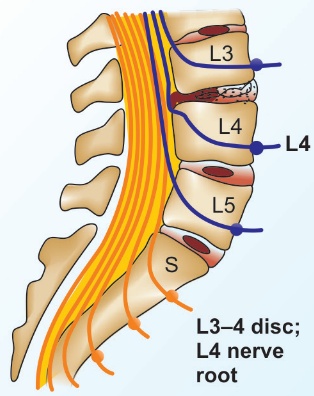
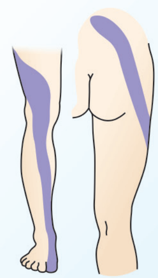
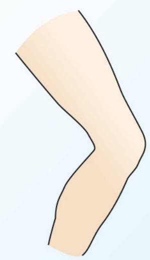
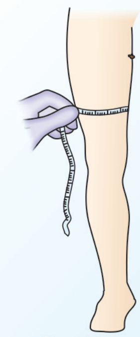
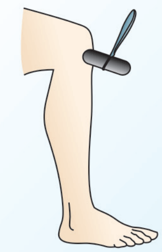
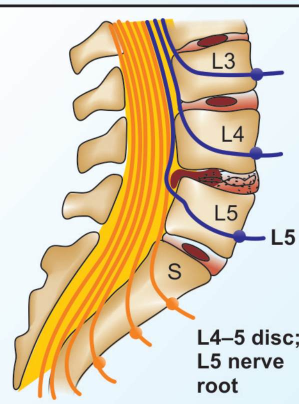
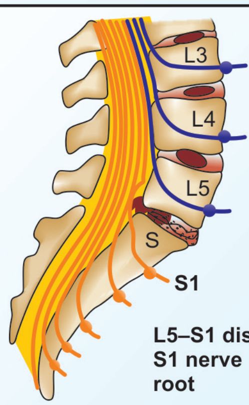
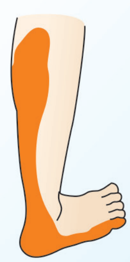
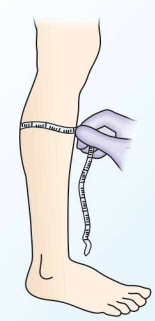
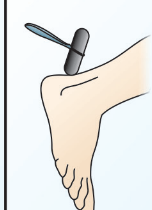
Clinical features					
Level of herniation	Pain	Numbness	Weakness	Atrophy	Reflexes
 L3–4 disc; L4 nerve root	 Lower back, hip posterolateral thigh, anterior leg	 Anteromedial thigh, knee and leg	 Quadriceps and knee extension	 Quadriceps	 Knee jerk diminished
 L4–5 disc; L5 nerve root	Just above sacro-iliac joint, hip, lateral thigh, and leg 	 Along lateral leg and first 3 toes	 Dorsiflexion of great toe and foot; difficulty in walking on heels; foot drop may occur	Not prominent	No change in knee and ankle jerk, Diminished or absent tibialis posterior reflex
 L5–S1 disc; S1 nerve root	Just above sacro-iliac joint, hip, postero-lateral thigh, and leg to heel 	 Along back of the calf, lateral heel, foot and toe	Plantar flexion of foot and great toe and difficulty in walking on toes	 Gastrocnemius and soleus	 Ankle jerk diminished or absent

Figure 11.4

Deficits observed at examination in intervertebral disc prolapse at various levels.

causing irritation and pain, the spinal column tilts laterally on the same side. The spine adopts this posture to prevent impingement and relieve compression and pain. If the extruded disc is lateral to the nerve root and compresses it, the spinal column tilts to the opposite side for the same reason as mentioned above. Therefore, the tilt of the spinal column indicates the position of the disc in relation to its nerve root. ***To summarize, the presence of a tilt to the same side as that of symptoms indicates that the compression is occurring medial to the nerve root and the presence of tilt to the opposite side as that of the symptoms indicates that the compression is occurring lateral to the nerve root (Fig. 11.5).***

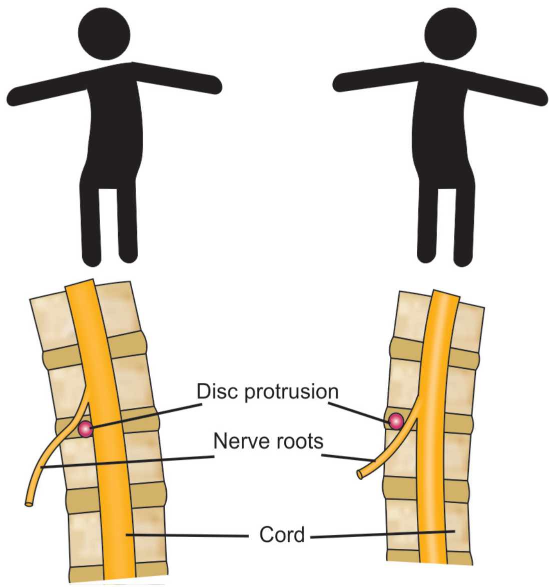


Figure 11.5

Medial and lateral position of the extruded disc in relation to the nerve root. To avoid irritation of the nerve root the spine tilts accordingly. To the same side when the extrusion is medial to the root and to the opposite side when the extrusion is lateral to the root. The patient develops a list.

Straight Leg Raising Test and Well Leg Raising Test

The 'straight leg raising test' (SLRT) is also known as the 'Lasegue's test' (1864).

With the patient lying down in supine position, lower limb on the affected side is raised with the knee in extension (Fig. 11.6). Normally, the leg can be raised to approximately 90° without radiating pain. This is said to be a negative test. A positive test is indicated by pain in the gluteal/hip region, radiating down to the leg along the course of the sciatic nerve.

The opposite lower limb when raised in a similar manner, produces pain on the involved side. This is known as a positive 'well leg raising test' or the 'Fajersztajn's test' (1901). The phenomenon is known as crossed sciatic phenomenon or sciatic paradox phenomenon.

The above tests are based on the principle of stretching the lumbar and sacral nerve roots. Cadaveric experiments have demonstrated that the nerve roots start moving towards the foramina with as low as 30° flexion of the hip. In a normal person, the range of excursion of the nerve roots is about 2–5 mm. In disc prolapse, when a leg raising test is performed, this excursion takes place over the protruded disc thereby causing irritation of the root and pain. The range of limitation in leg raising is directly proportional to the severity of the disc prolapse. For example, a positive test at 30° leg raising indicates more severe degree of disc prolapse than a positive test at 60°.

Figure 11.6

'Straight leg raising test' being performed. In this it is negative and the leg can be raised up to 80°.

In a positive well leg raising test which is positive in about one-third of the cases of intervertebral disc prolapse, it has been found that the protruded disc is in the axilla or medial to the opposite root, and never lateral.

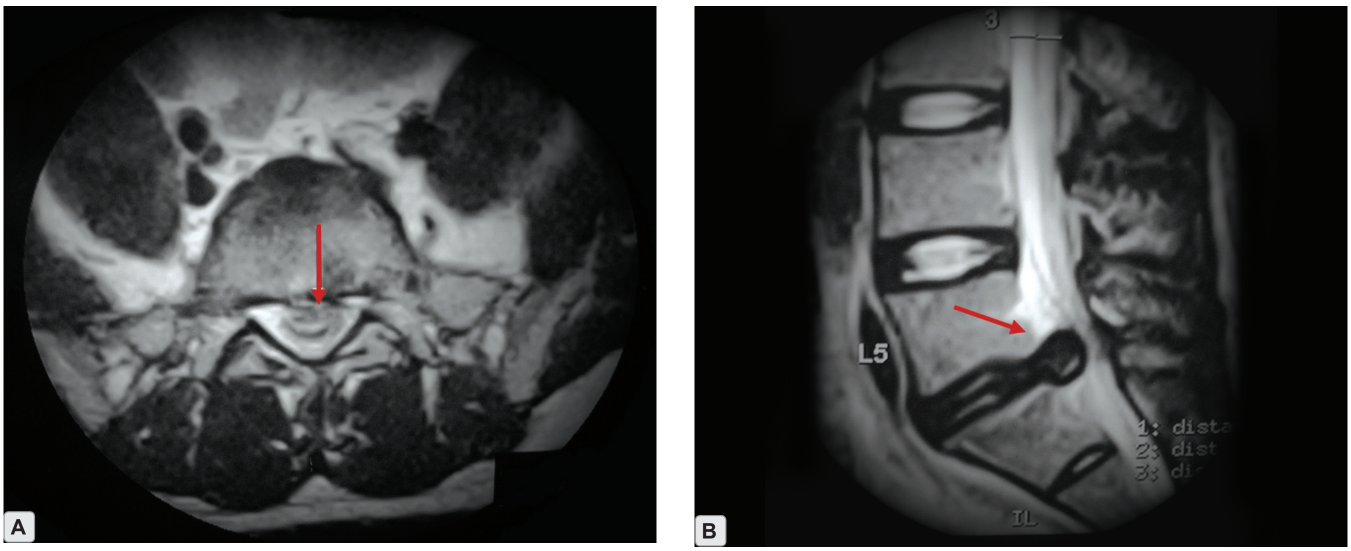
Note: *The symptoms can be exaggerated by dorsiflexing the ankle or by asking the patient to flex the neck. These maneuvers stretch the nerve root further and help in re-asserting a positive test or recognizing an early prolapse when SLRT is inconclusive.*

Investigations

X-ray may show loss of normal lordosis and presence of scoliosis. In cases of degenerative disc it shows changes of degeneration with osteophyte formation in the spinal column.

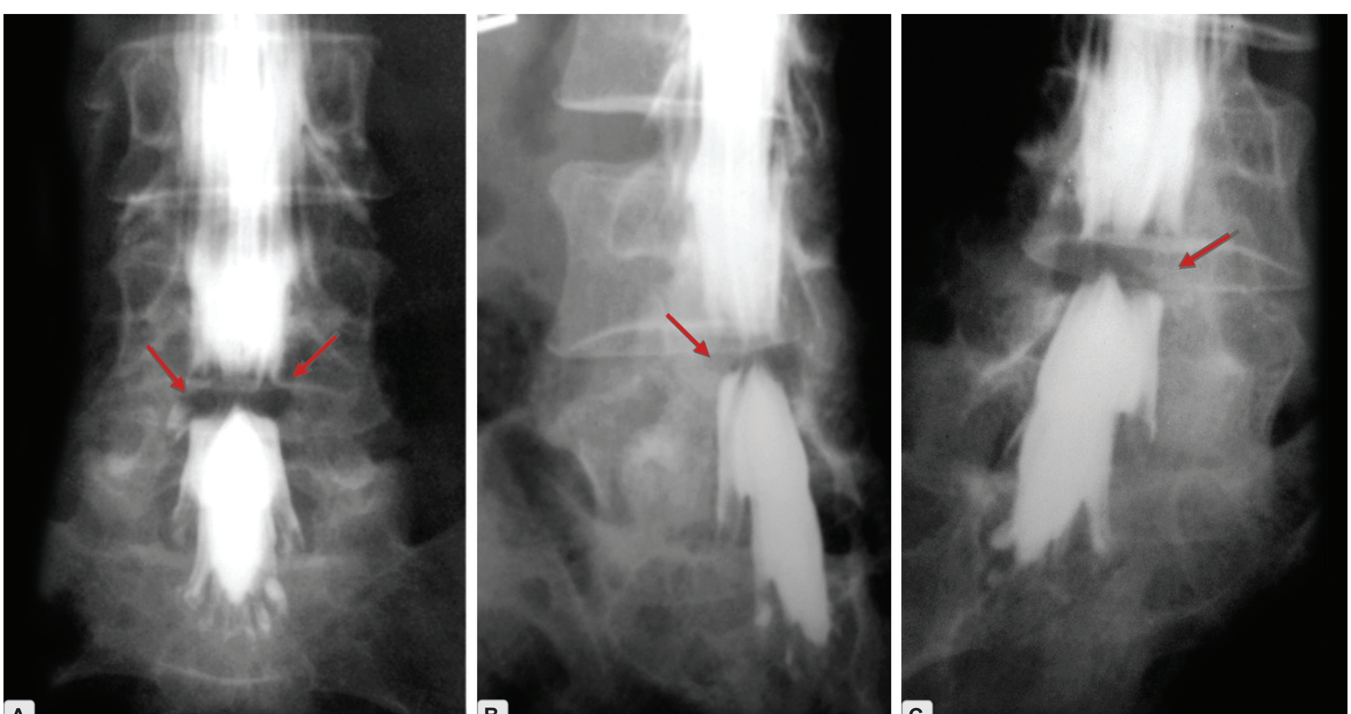
Magnetic resonance imaging (MRI) is the investigation of choice. It clearly shows the nature and extent of prolapse and the site of nerve root compression (Figs 11.7A and B).

Myelogram was the standard investigation for intervertebral disc prolapse before MRI was invented. Because of its invasiveness, the risks of spinal puncture with side effects and complications of the dye used, it has been given up these days. The picture of myelogram is used here (instead of diagrammatic representation) to show the exact nature of nerve root involvement in IVDP (Figs 11.8A to C).



Figures 11.7A and B

MRI showing prolapsed and extruded intervertebral disc at L5 S1 compressing the S1 root.



Figures 11.8A to C

Myelogram showing a midline disc herniation L4–L5 with bilateral L5 root cut off.

Treatment

The intervertebral disc prolapse can be treated nonoperatively or operatively. The decision is always taken in consideration with clinical features and MRI findings.

Nonoperative Management

It is indicated in those cases with no or minimal neurological symptoms and MRI shows only protrusion and not extrusion.

Following are the measures taken:

a. Bed rest and immobilization in traction.
b. Anti-inflammatory drugs.
c. Lumbosacral corsets for support during the period of gradual mobilization.
d. Spinal strengthening exercises to follow.
e. Advise to abstain from such activities which impart loading stress on the spine, e.g. lifting heavy weights, uncomfortable travel, poor posture at work, etc.

Operative Management

It is indicated when the symptoms and neurological deficits are severe and MRI shows extrusion or sequestration of the disc. Earlier the decompression of the root, faster is the relief and better is the recovery.

Procedures:

a. Laminectomy and discectomy
b. Hemilaminectomy and discectomy
c. Fenestration/laminotomy and microdiscectomy
d. Endoscopic discectomy

Among the above procedures indications for a and b procedures are few. More commonly c and d procedures are preferred and done routinely because they are less invasive.

Chemonucleolysis: In this procedure, the protruded *disc* which is symptomatic is *dissolved by injecting the enzyme chymopapain* derived from the papaya, into the disc space. The technique needs expertise and skill. *On table discogram* is a must. If the *dye leaks out into the epidural space the procedure is abandoned.* The procedure is *also abandoned* if there is *dural puncture and spinal fluid leak.*

Anaphylaxis due to allergy to the enzyme and *late development of transverse myelitis* and its neurologic sequel are the risks involved. The procedure is rarely done these days.

Artificial disc replacement: Artificial disc replacement is being done in those patients with degenerated discs who have disabling back pain, who have not responded to nonoperative line of management and in whom arthrodesis is the option. The artificial disc is designed with ultrahigh molecular polyethylene core, sandwiched between cobalt chromium endplates and aims at restoring pain free movement at that level. Centers where these surgeries are being done have claimed successful results in the range of 60–90% at 7 years follow-up.

Problem of degeneration of posterior elements and facetal joints, the polyethylene wear and the effect of its debris on the spine and the cord and the salvage procedures adopted when the implant fails, are some issues which are being closely studied at this point of time.

Revision Questions

Q. Write notes on:
   a. SLRT
   b. Well leg raising test.
   c. List
   d. Sciatic scoliosis
   e. Disc prolapse.

Q. Write briefly on:
   a. Laminectomy
   b. Hemilaminectomy
   c. Fenestration
   d. Microlumbar discectomy.

Essay Question

Q. Discuss the etiopathogenesis, clinical features, diagnosis and management of a case of acute intervertebral disc prolapse. Enumerate its complications.

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12 Nutritional, Endocrine, Degenerative and Autoimmune Disorders

- *Rickets and osteomalacia*
- *Osteoporosis*
- *Rheumatoid arthritis*
- *Hyperparathyroidism*
- *Osteoarthritis*

RICKETS AND OSTEOMALACIA

Rickets and osteomalacia are abnormalities resulting in lack of mineralization of the bone due to deficiency of vitamin D. Vitamin D is the name given for a group of sterol molecules. The two important forms of vitamin D are vitamin *D2 otherwise known as ergocalciferol* derived from plants and *D3 otherwise known as cholecalciferol* derived from animals, e.g. egg yolk, cod liver oil, etc. Cholecalciferol is also synthesized in the skin of animals from 7-dehydrocholesterol by exposure to sunlight. *Cholecalciferol i.e. Vitamin D3 is converted into biologically active form by a process of hydroxylation which occurs first in the liver and then in the kidneys, respectively.*

In the liver, the enzyme 25-hydroxylase converts the cholecalciferol into 25-hydroxycholecalciferol/Calcidiol.

In the kidneys, the enzyme 1-alpha hydroxylase converts the 25-hydroxycholecalciferol into 1,25-dihydroxycholecalciferol/Calcitriol (Fig. 12.1).

This hydroxylated active form of vitamin D is important for regulation of calcium and phosphorus levels in the blood and for mineralization of bone.

Biologically active vitamin D is fat-soluble and is transported through the bloodstream by the help of a binding protein. 1,25-dihydroxyvitamin D has a half-life of a few hours. Whereas 25-hydroxyvitamin D has a half-life of several weeks. *The liver acts as a storehouse of vitamin D. It stores the vitamin D synthesized in the skin and absorbed from the intestines, in 25-hydroxylated form and releases it into the bloodstream as per requirement.*

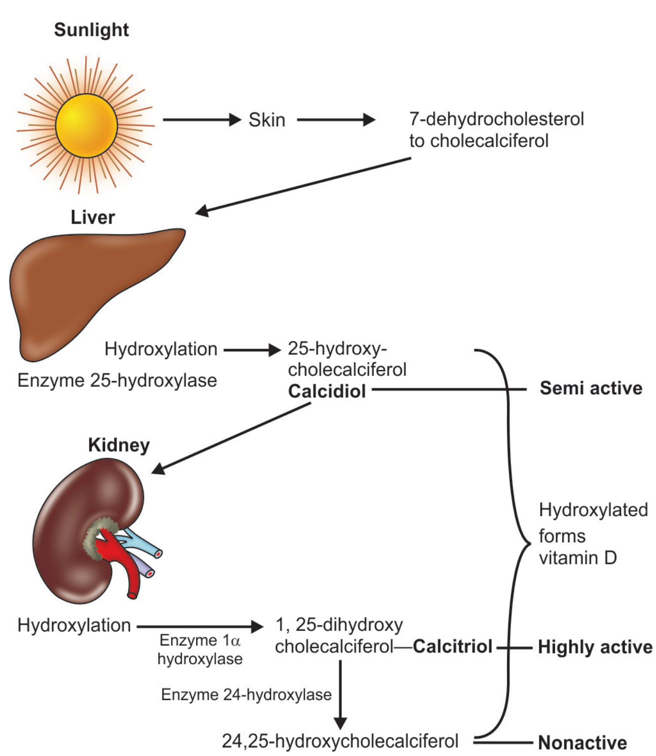


Figure 12.1
Synthesis of vitamin D and its activation.

Once the calcium and phosphorus level is regulated the formation of 1,25-dihydroxycholecalciferol stops and formation of nonactive 24,25-hydroxycholecalciferol begins. Thus Calcitriol has an autoregulatory influence on its synthesis in the kidneys.

Note: *Cod liver oil, Shark liver oil, Halibut liver oil are some of the richest sources of vitamin D. The polar bear liver is the richest source of vitamin D among animals. It is so toxic that ingestion results in death in humans. Hence, Eskimos do not eat the liver of the polar bear. The toxic property of vitamin D is made use of in preparing rodenticide. Baits smeared with vitamin D are available as rodenticide.*

Rickets

Etiology and Classification

Rickets can be classified based on etiology as nutritional, renal, etc. as well as depending on the levels of calcium and phosphorus in the blood as calcipenic or phosphopenic. While nutritional rickets is always calcipenic, rickets due to genetic causes can be calcipenic or phosphopenic (Flow chart 12.1).

A. *Nutritional:* Dietary deficiency of vitamin D, calcium and phosphorus or intestinal malabsorption of calcium results in this form of rickets. The condition is reversible when attention is given to the diet. Not sex-linked and no inheritance is observed.

B. *Lack of exposure to sunlight:* 7-dehydrocholesterol is not converted into cholecalciferol. This disturbs the process of hydroxylation in the liver resulting in deficiency of Calcidiol.

Both A and B are examples of 'nutritional, calcipenic rickets'.

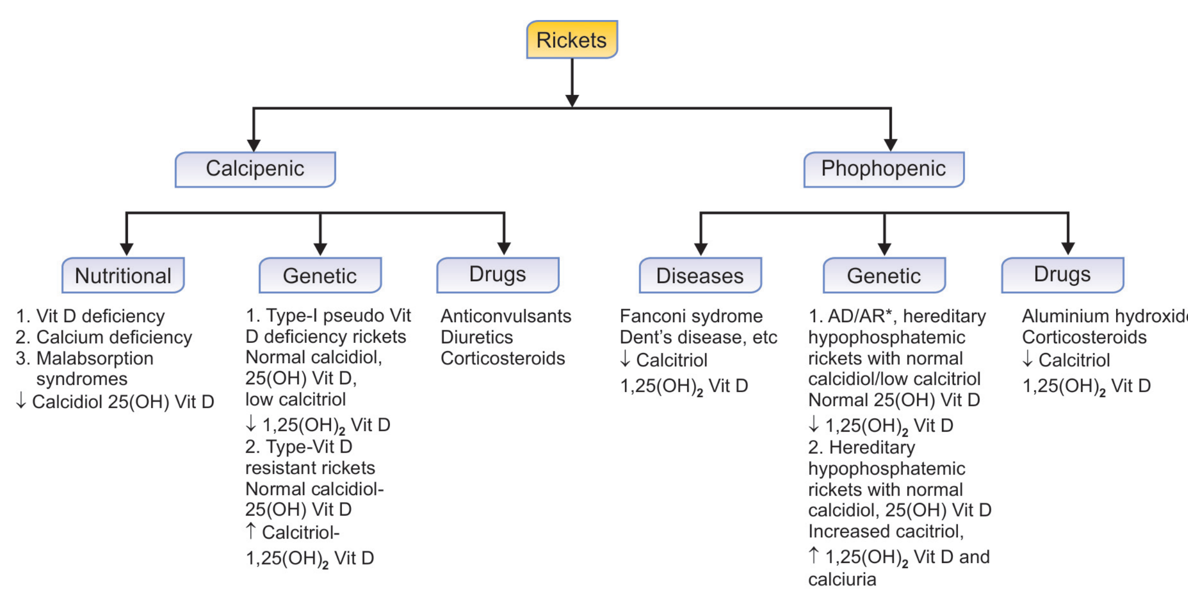
C. *Genetic causes*: Vitamin D-dependant rickets

  a. Type I—In this type there is *deficiency of the enzyme 1-alpha hydroxylase* in the kidneys. Thus *deficiency of 1,25-dihyroxyvitamin D* (Calcitriol) *develops.* Autosomal recessive inheritance is observed. It is also known as pseudovitamin D deficiency rickets. Supplementation with active form of vitamin D, i.e. Calcitriol (Vitamin D_3) results in cure.

  b. Type II—In this type the *production of 1,25-dihydroxyvitamin D is normal.* But the *receptor is abnormal.* Hence, there is defective interaction between the vitamin D and the receptor *resulting in resistance.* Increased level of Calcitriol in the blood helps in the diagnosis. Autosomal recessive inheritance is seen. High

Flow chart 12.1

Classification of rickets.
In calcipenic rickets,
↓serum calcium, N/↓serum phosphorus, ↑parathormone, ↑alkaline phosphatase
In phosphopenic rickets,
N serum calcium,↓serum phosphorous, N parathormone, ↑alkaline phosphatase.



* AD-Autosomal dominant
AR-Autosomal recessive
N-Normal

levels of vitamin D in the blood is necessary for control. This type of rickets cannot be cured but can be controlled with large doses of vitamin D. Hence supplementation with large doses of vitamin D may become necessary depending on serum levels of Calcitriol. This is known as Vitamin D resistant rickets. Both Type—I and Type—II rickets are examples of 'genetic, calcipenic, rickets'.

Hypophosphatemic rickets:

i. Familial—In this variety there is renal tubular abnormality which leads to decreased re-absorption of phosphates. This results in *phosphaturia and hypophosphatemia.* X-linked dominant inheritance is observed. *Calcitriol levels are low.* Hence, need supplementation of both, calcitriol and phosphates.

ii. Hereditary—In this variety there is *hypercalciuria, phosphaturia and hypophosphatemia* because of decreased renal tubular reabsorption of phosphates. But the serum levels of *calcitriol are high.* Hence, needs supplementation of phosphorus only. Autosomal dominant/recessive inheritance is seen.

Both the types of hypophosphatemic rickets are examples of 'genetic, phosphopenic rickets'.

D. Chronic renal and liver diseases.
Renal osteodystrophy, cirrhosis, coeliac disease etc. In these conditions hydroxylation process is interfered. Hence active forms of vitamin D are not available in the blood. The resultant malabsorption causes calcipenic rickets.

E. Others
Oncogenic, drug induced (phenytoin, antacids containing aluminum), etc.

Differences between nutritional rickets and vitamin D resistant rickets

Nutritional	*Vitamin D resistant*
Acquired	Inherited
Responds to adequate doses of vitamin D	Responds only to large doses of vitamin D. Supplementation of phosphorus may be necessary
Serum chemistry becomes normal following vitamin D administration	Serum chemistry remains abnormal even after administration of large amounts of vitamin D
Spontaneous resolution of deformities is possible with vitamin D	Spontaneous resolution of deformities is not possible even with large doses of vitamin D
Growth becomes normal on healing	Growth never becomes normal Always remains stunted

Pathology

Pathology is defective mineralization. *In children*, both the growth plate and bone get affected. Hence, skeletal changes of both *rickets and osteomalacia develop.* Whereas *in adults,* vitamin D deficiency causes changes of *osteomalacia only because the growth plate is already fused.*

In the growth plate, the stages of bone formation proceed normally up to the stage of appearance of mature chondroblasts leading to chondrocyte formation and laying down of cartilaginous matrix. At this point, the process of calcification becomes deficient. This interferes with the normal progress of growth. As a result, poorly mineralized osteoid formation occurs in the physis with accumulation of nonossified cartilage. This is responsible for the classical radiological picture of widening of the physis and cupping of the metaphysis.

In the diaphyseal bone, in children as well as in adults, the mineralization of the osteoid does not take place. Hence, osteomalacic changes develop.

Thus, bones become soft because of lack of mineralization and bend. This results in classical deformities, i.e. bow legs, knock knees, windswept deformity, etc. Pathological fractures also can develop.

Clinical Features

Rickets

Child may present with following features (Fig. 12.2):

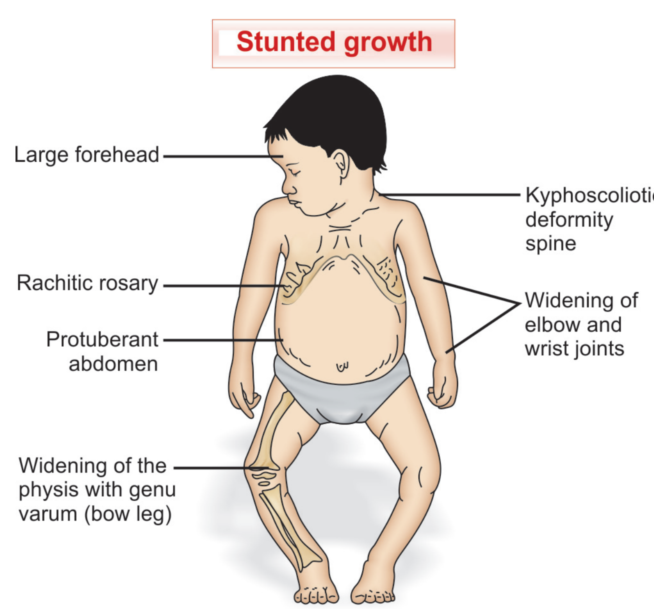


Figure 12.2
Child showing features of rickets.

General

- Irritable child
- Lethargy and hypotonia
- Pot belly
- Delayed milestones
- Stunted growth

Specific

- Craniotabes (soft skull).
- Bossing of the skull.
- Pigeon chest.
- Rachitic rosary/rickety rosary (Costochondral beading).
- Harrison's groove—is a line observed at the place where the diaphragm attaches to the ribs.
- Bowing or widening of the physis.
- Deformities in the lower limbs, e.g. genu valgum, genu varum, windswept deformity (Figs 12.3B and C).
- Deformities in the spine—kyphoscoliosis.
- Radiologically, osteopenia with widening of the physis and cupping of the metaphysis (Figs 12.4A to F).
- Pathological fractures due to softening of the bones.

Osteomalacia

- Nonspecific features in early stages.
- Bone pain may be the presenting feature.
- Muscular weakness and easy fatigue.
- Carpopedal spasm and facial twitching.
- Pathological fractures (commonly seen in the spine).

Assessment of Bilateral Genu Varum and Genu Valgum

Genu varum: When the intercondylar distance, i.e the distance between the two condyles of the femur, increases (approximately > 4 inches/10 cms) it indicates bilateral Genu varum.

Genu valgum: When the intermalleolar distance, i.e. the distance between the two malleoli of the tibia, increases (approximately > 4 inches/10 cms) it indicates bilateral Genu valgum.

Investigations

Investigations are done to confirm the type of rickets for adequate planning of treatment.

Nutritional Rickets

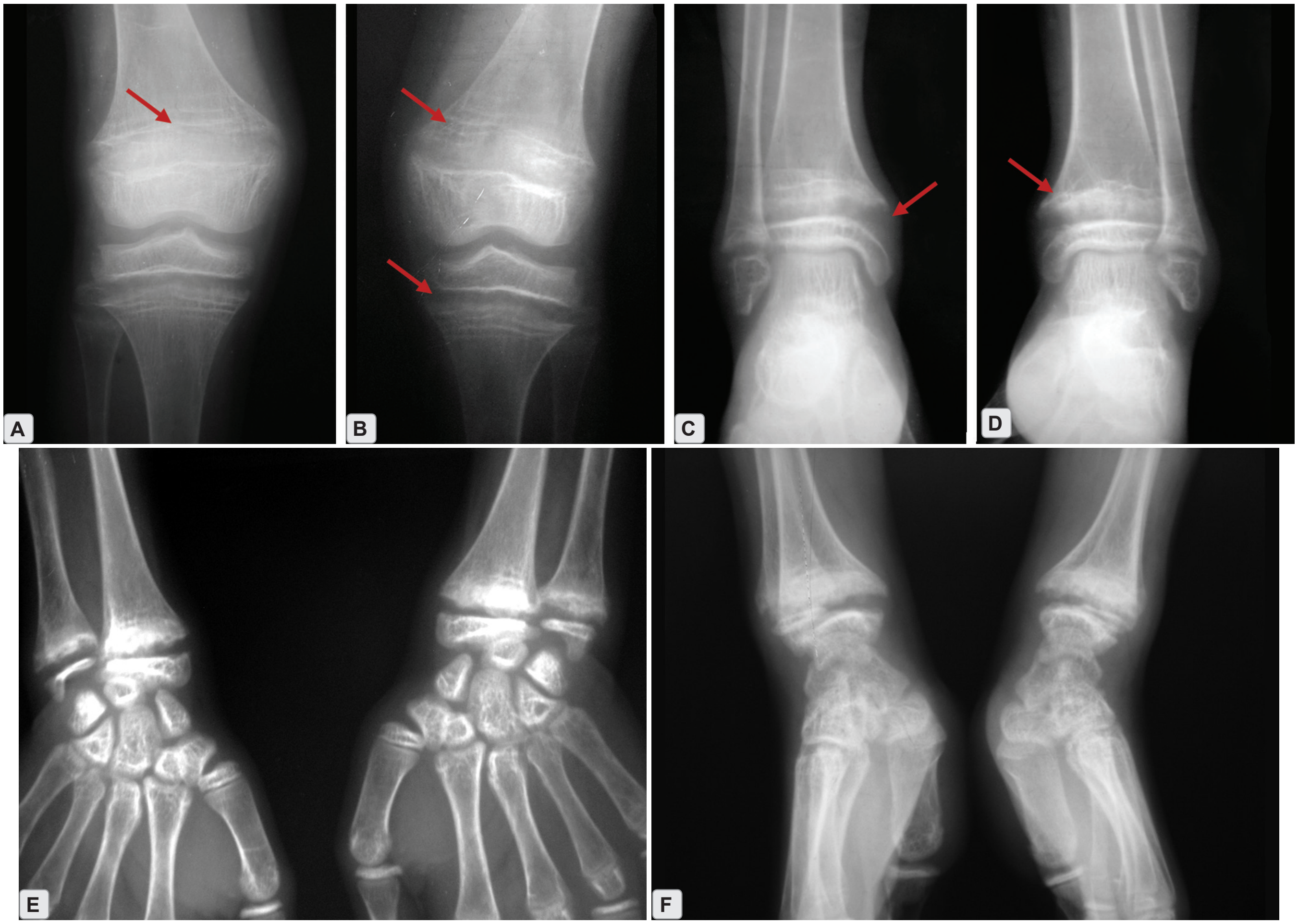
a. Serum calcium, phosphorus and alkaline phosphatase. Serum calcium and phosphorus is low, alkaline phosphatase is elevated.
b. Urinary calcium is low.
c. Serum calcidiol and calcitriol is low.
d. PTH is elevated.
e. Urinary phosphorus is elevated.

Vitamin D Resistant Rickets (Type II)

a. Urinary calcium may increase.
b. Serum calcitriol levels may show an increase.

Figures 12.3A to C

Cupping of the metaphysis, widening of the physis and genu valgum and windswept deformities: (A) Widening of the wrist and trumpeting of the metaphysis; (B) Knock knee; clinical and radiological picture (C) Windswept deformity: Bow leg on one side and knock knee on the other.



Figures 12.4A to F

Classical changes of rickets at the ends of long bones. Note the presence of Harris growth lines in the metaphyseal portion of the bone which indicate intermittent growth retardation (marked by red arrow). These lines are named after Henry Harris a Welsh anatomist. They are seen in children when temporary growth retardation takes place.

X-rays Changes in Rickets

Classical changes of *widening of the physis and cupping of the metaphysis* is seen (Figs 12.3A and 12.4A to F). These changes are more pronounced in the region of the knee and the wrist. Further, rarefaction of the bone is observed confirming *osteomalacic changes.*

Skull may appear square, known as *Caput Quadratum.*

X-rays Changes in Osteomalacia

- The skeleton looks thinned out and casts a poor radiological image.
- Looser's zones may be seen. These are areas of pseudofractures occurring at the sites of stress. Pubic rami, medial cortex of neck of femur, axillary border of the scapula are some of the sites for pseudofractures. These are the result of increased rate of bone resorption and poor new bone deposition.
- Protrusio acetabuli may be seen in the pelvis.

Treatment

Nutritional Rickets

a. Adequate exposure to sunlight (not excessive exposure)
b. Supplementation of vitamin D in the diet.

} Preventive and curative measure

(Recommended even for breast-fed babies after two months of age)

c. Treatment with therapeutic doses of vitamin D 200–600 units/day or as a intramuscular injection of 600,000 IU. Which may have to be repeated at 4–6 weekly intervals.

Radiological sign of healing: A sclerotic line developing in the metaphyseal region (Figs 12.5A to C).

In refractory rickets this does not occur.

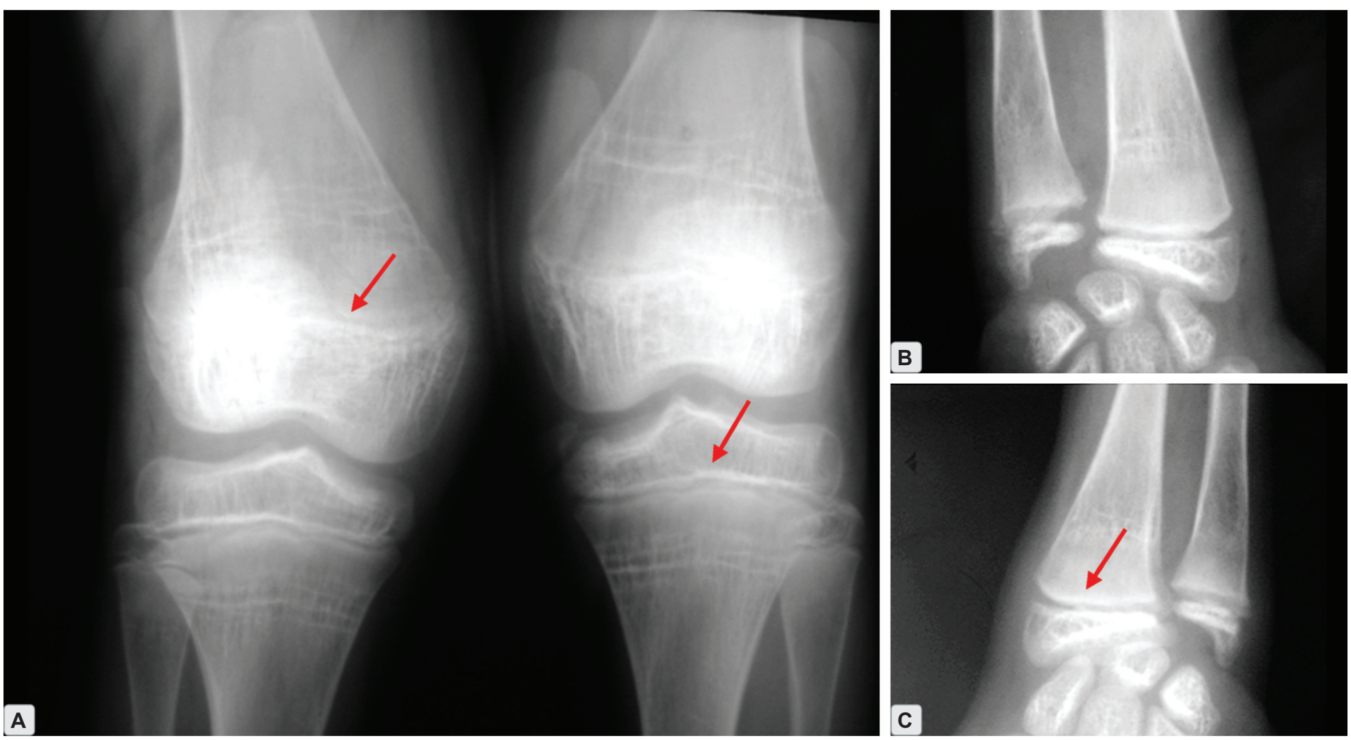
Familial and Hereditary Rickets

These are refractory to usual treatment because of genetic abnormality. Depending on the cause as discussed in the etiology, the treatment varies. Large doses of vitamin D and supplementation of phosphorus may be necessary and the treatment may last life long.

Correction of Deformities in Rickets (Figs 12.6A and B)

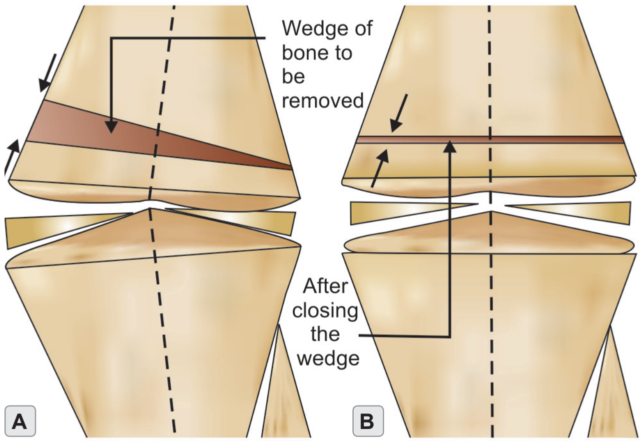
In young, the deformities get corrected with adequate supplementation of vitamin D and splinting. Deformities which are of severe nature and those which are established need correction. Correction is always done when the serum parameters become normal after treatment with vitamin D.

The procedure of corrective osteotomy is undertaken to correct the deformities, e.g. Mac Evan's osteotomy (medial close wedge osteotomy) for genu valgum.



Figures 12.5A to C

Same patient shown in Figures 12.4A to F after adequate vitamin D administration. Complete healing is seen as evidenced by the presence of sclerosis and physeal width becoming normal. Note the presence of Harris growth lines in the metaphysis which indicate intermittent growth arrest.



Figures 12.6A and B

Mac Evan's medial closed wedge osteotomy for the correction of genu valgum. Note the angle of valgus deformity. This angle decreases after taking an appropriate wedge of bone with the base directed medially. The defect is closed after removing the wedge of bone. The size of the wedge depends on the extent of correction required.

Revision Questions

Q. Write notes on:
   a. Active forms of vitamin D
   b. Actions of vitamin D
   c. Rickets
   d. Osteomalacia
   e. Radiological picture of rickets
   f. Vitamin D resistant rickets.

Essay Question

Q. Classify rickets. Discuss the clinical features, diagnosis and management of different types of rickets.

OSTEOPOROSIS

Osteoporosis may be defined as a generalized disorder involving the skeleton in which the bone density decreases, the fragility of bone increases and as a result the bone becomes brittle and more susceptible for fractures.

Types

a. Postmenopausal (Estrogen lack)
b. Senile (Natural process)

Risk Factors

a. Advancing age
   Women > 45 years (after menopause); Men > 60 years
b. Low calcium and vitamin D in the diet
c. Lack of exercise
d. Smoking
e. Drug-induced, e.g. long-term steroids

Pathology

The strength of the bone depends on proteins, collagen and calcium. This inherent strength is acquired genetically *in utero* itself influenced by nutritional, hormonal and environmental factors. The influence of these factors on bone mass continues throughout life.

Bone is a dynamic structure. Throughout life, there is a continuous process of new bone deposition, bone resorption and remodeling. In the normal course the deposition and resorption of bone balance each other and are responsible for maintaining the standard quality of the bone. When the resorption of bone exceeds that of deposition thinning of bone occurs and osteoporosis develops, i.e. osteoclastic activity > osteoblastic activity. The osteoclastic activity is regulated by RANK ligand and the osteoblastic activity is regulated by osteoprotegerin (OPG). RANK and OPG have opposite effects on the bone.

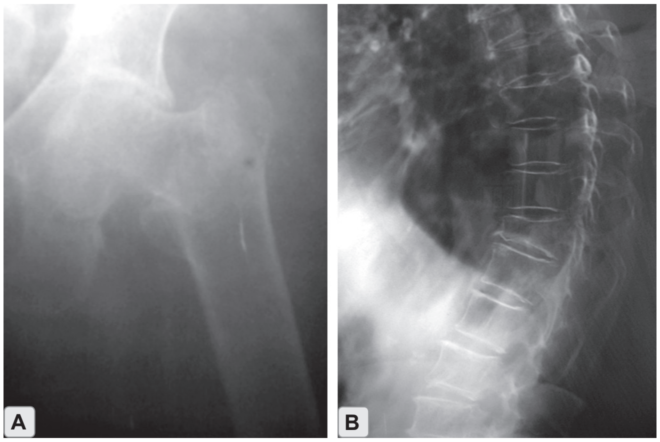
Clinical Features

Osteoporosis does not give rise to any clinical symptoms on its own. Only when a fracture occurs symptoms develop (Fig. 12.7A) and a patient starts complaining of pain and inability to move the part. Generally the injury is very trivial. The fracture can be incomplete or complete. In the spine, compression fracture is common and patient presents with backache with/without radiating pain. If the fracture involves the pelvic bone, the patient complains of pain in the groin and difficulty in walking.

Investigations

X-ray shows thinning of the bony trabeculae and generalized rarefaction.

In the spinal column, osteoporotic compression fractures may be seen in the vertebrae (Fig. 12.7B).



Figures 12.7A and B

(A) Fracture in the intertrochanteric region and (B) a wedge compression of T-12 vertebra due to osteoporosis. The radiographs show marked thinning of both cortical and cancellous bones.

Bone densitometry by DEXA (Dual energy X-ray absorptiometry) is more precise and can detect osteoporosis early.

Treatment

Drug Therapy

The therapy should be instituted when risk factors exist, especially after 45 years of age, after confirming the diagnosis.

Aim: Is to prevent osteoporotic fractures.

Two basic types of drugs are used to treat osteoporosis.

a. Drugs that inhibit the osteoclast-induced bone resorption, e.g. Risedronate.
b. Drugs that promote osteoblastic induction of bone, e.g. Teriparatide.

Along with these drugs calcium and vitamin D are also given in adequate doses.

Recent drug is a stable strontium compound. Strontium renolate has dual action of increasing bone synthesis and decreasing bone resorption. Monoclonal antibodies are also being developed by recombinant technique. For example, Denosumab. It binds with the RANK-Ligand, which otherwise would have bound with RANK receptor on osteoclast and induced osteoclast activation. Hence, in future there may be two more additional drugs—dual acting bone agents (DABAs) and Monoclonal antibodies.

Treatment of Fractures

- Vertebral fractures involving *single vertebra* can be managed by *vertebroplasty* using injection of bone cement. This is followed by a protective brace at least for three months.
  During this period it is advisable that the patient does not lift any heavy object.
- *Long bone fractures* are problematic to treat. They have to be treated by reduction and *internal fixation* followed by protective immobilization. In severely *comminuted osteoporotic fractures*, *bone grafting may be necessary.*
- Appropriate drug therapy is a must.

Essay Question

Q. Discuss the etiopathogenesis, clinical features, diagnosis and management of senile osteoporosis. What are the complications of senile osteoporosis?

OSTEOARTHRITIS

Degenerative arthritis, hypertrophic arthritis, chondromalacic arthrosis and osteoarthrosis are the other names for osteoarthritis. The condition is the result of degenerative changes taking place in the joint. The articular cartilage and the subchondral bone progressively degenerates. This results in inflammation and mechanical derangement which promotes further degeneration. Ultimately results in badly deranged joint, associated with deformity and ligamentous laxity.

Types

a. *Primary osteoarthritis:* In this, there is no pre-existing abnormality or damage (incongruity) for causing progressive degeneration.
b. Secondary osteoarthritis: Here, there is a pre-existing pathology or damage (incongruity) which is responsible for the progressive degeneration.

Note: *The basic difference in joint destruction occurring in rheumatoid arthritis and osteoarthritis is that, in rheumatoid arthritis, inflammation occurs first. The joint destruction that follows is the result of inflammation. Whereas in osteoarthritis, the joint destruction occurs first and the inflammation that follows is secondary to joint destruction.*

Etiology and Pathogenesis

Exact etiology is not yet understood. But, certain factors predispose to the development of osteoarthritis.

a. *Trauma:* Minor degrees of repetitive trauma occurring during activities of daily living causes osteoarthritis. There is a protective mechanism operating in the human body when it is subjected to unprepared stress. For this reflex protective mechanism to operate, it takes a few milliseconds, e.g. when there is a slip. The injury is avoided if the reflex protective mechanism operates by contraction of different muscles which in turn averts a fall. But, those repetitive minor stresses (trauma) occurring within a few milliseconds before the reflex protective mechanism operates, causes injury to the joint structures. This (perhaps a microfracture) is thought to be responsible for most of the degenerative arthritis, e.g. osteoarthritis of the finger joints occurring in musicians and typists, cervical spondylosis occurring in head load workers and sky divers of Acapulco in Mexico. (La Quebrada cliff where sky divers dive into the sea from a height of about 45 meters). Osteoarthrosis of the knee is common among sailors and people who stand for long hours at work.
b. *Genetic factors*: Osteoarthritis is known to occur among many family members. It is especially common in identical twins. These factors support genetic predisposition.

Pathology

Primary

With advancing age the proteoglycan content of the articular cartilage decreases thereby reducing the water content of the cartilage. Such a cartilage becomes less resilient and cracks during activities of daily living. This is responsible for the onset and progression of degenerative process. It starts innocuously in the beginning, progresses slowly over a period of time and then becomes severe (Fig. 12.8).

Secondary

There is a joint incongruity which has developed due to some disease in the past. Thus, degeneration occurs secondary to a pre-existing mechanical abnormality.

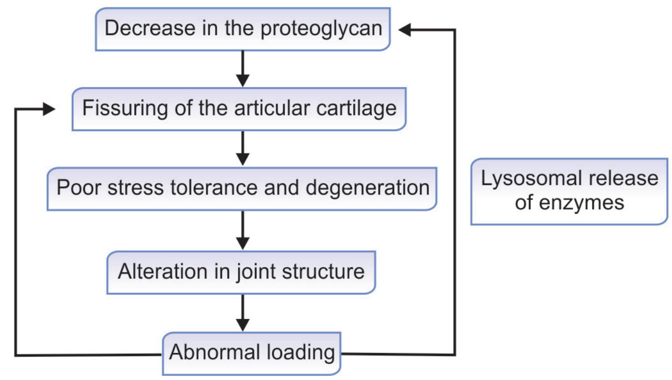


Figure 12.8
Vicious cycle of idiopathic degeneration in primary osteoarthritis.

This is secondary osteoarthrosis (See Chapter 10 Figs 10.15A and B).

Clinical Features

a. *Pain:* It is the primary feature of osteoarthritis. It is gradual in onset and slowly progressive in severity. There can be pain free intervals also.
b. *Swelling and joint effusion:* Swelling in osteoarthritis is due to effusion. This is commonly seen early in the course disease. Whereas, late in the course of the disease both swelling and deformity are seen.
c. *Limitation of movement:* Painful limitation of movement is a late feature of osteoarthritis. In the early phase the joint movements are associated with pain but not limited.
d. *Heberden's nodes and Bouchard's nodes:* These are nodules occurring in the DIP and PIP joints respectively, in osteoarthritis.

Diagnosis

It is not difficult. Clinical features and X-rays confirm the diagnosis.

Radiological Findings (Figs 12.9A to D)

- Narrowing of the joint space (due to loss of articular cartilage).
- Sclerosis of the subchondral bone.
- Subchondral cysts.
- Osteophytes (the bony excrescence or outgrowth, usually branched in shape).
- Deformities.

Figures 12.9A to D

A 60-year-old patient with severe osteoarthritis. Note the extensive degenerative changes suggestive of eburnation of the articular surface. Osteophytes are seen in plenty. Joint space is narrowed and there is evidence of instability as seen by opening up of the lateral joint space. This patient underwent total knee replacement and is totally symptom free.

Management

Nonoperative Management

In early stages of the disease, the treatment aims at providing pain relief and slowing down the pace of progression of the disease by taking care of the offending factors if any, e.g. overweight and giving exercise therapy to strengthen the muscles. A good functioning muscle acts as a shock absorber and takes care of the stress which otherwise would have fallen maximally on the joint.

Nonsteroidal anti-inflammatory drugs take care of the pain and inflammation. Drug therapy along with good physiotherapy, gives considerable amount of pain relief.

Infiltration of hyaluronidase into the joint is known to give sustained temporary pain relief.

Surgical (Operative) Management

It is done late in the course of the disease when the joint is degenerated and clinically associated with pain and deformity. Total joint arthroplasty is the treatment of choice (Figs 12.9A to D).

In selective cases, osteotomies can be done. Osteotomy alters the line of weight bearing and distribution of stress and is known to give relief, e.g. high tibial osteotomy.

Arthroscopic lavage and excision of loose bodies is done in high-risk patients who are not fit for joint replacement surgery in case of Osteoarthritis of the knee. This procedure gives temporary relief of symptoms.

Essay Question

Q. Discuss the etiopathogenesis, clinical features, radiological features, diagnosis and management of osteoarthritis.

RHEUMATOID ARTHRITIS

Definition

It is an autoimmune systemic disorder wherein the body defence mechanism identifies the normal tissues as a non self antigen and evokes a chain of inflammatory reaction resulting in destruction.

Etiology

Exact cause is not known. Following factors are considered as triggering factors.

a. Chronic infection—bacterial, fungal or viral.
b. Environmental factors—polluted atmosphere, smoking, etc.
c. Genetic factors—HLA with DR4 specificity is observed.
d. Endocrinal factors.
e. Poor quality food with constant assault on the gut.

Joints, Organs and Tissues Involved

Mainly involves the joints. Joints of the wrist, hand, knee, ankle and foot are commonly affected. Elbow, shoulder, hip and spine are not commonly affected. Monoarticular involvement is rare.

Involvement of soft tissue structures surrounding the joint also occurs, e.g the synovium, capsule, tendons, muscles, etc.

Lungs, pericardium, eyes, vessels and nerves are the other structures that can get involved.

- Age 20–40 years.
- M:F ratio is 1:3.

Pathology

The disease process starts in the synovium as synovitis with infiltration by inflammatory cells; to begin with monocytes infiltrate the synovium. This is later followed by lymphocytes. As a result, the synovium gets thickened. An effusion into the joint develops and as the disease progresses, the cytokines released in the process, e.g. TNF-α, interleukin-1 and interleukin-6, histamine, etc. destroy the articular cartilage, the bone, the capsule, the ligaments, the tendons and the muscles. The destruction occurs by the formation of a pannus of tissue at the junction of the synovium and the articular cartilage. This pannus erodes the cartilage and the bone and destroys it. Thus, arthritis develops.

Clinical Features

- Systemic features start insidiously with low grade fever, malaise, fatigue and joint pains.
- The joints are swollen edematous, warm, tender with effusion and the movements are painfully limited. Deformities may develop in the joints (Fig. 12.10A).
- Early morning stiffness is characteristic.
- Synovial thickening is appreciable.
- Subcutaneous nodules may be palpable.

Investigations

Blood Investigations

Hb%—May be low.

TC—Leukocytosis.

DC—Neutrophil increase in the early acute phase and in chronic phase may show increase in lymphocytes.

ESR—Elevated.

CRP—Elevated. It is nonspecific and only suggests that there is active inflammation.

ANA—When positive suggests sensitization has occurred and autoimmunity has developed.

(Normal titer is 1:40. Higher titer indicates presence of autoimmunity).

Rheumatoid/RA factor—Positive in 80% of the patients. > 20 IU/ml is considered as positive.

CCP (Citrulline antibody)—Elevated. It is more sensitive than Rheumatoid factor. Can be positive even when Rheumatoid factor is negative.

What is ANA (Antinuclear Antibodies)?

Antinuclear antibodies are autoantibodies. These have a capability of binding to the nucleus of a normal cell, which it recognizes as an antigen and makes it vulnerable for destruction.

Figures 12.10A to C

(A) Classical swan neck deformity due to tightness of the intrinsic muscles as a result of inflammation and fibrosis; (B and C) Ankylosis of the wrist joint in a burnt out case of rheumatoid arthritis. Note the extensive destruction that has occurred.

What is Rheumatoid Factor?

Rheumatoid factor is an autoantibody against the Fc portion of IgG. It is seen in about 80% of the patients with rheumatoid arthritis. It is not 100% specific. False-positive results are possible.

Hence, the result is to be correlated with clinical symptoms.

What is CCP Antibody?

CCP stands for cyclic citrullinated peptide (CCP) antibody. Citrulline antibody titer is a better diagnostic test than 'Rheumatoid factor'. When elevated, it not only helps to diagnose rheumatoid arthritis but also indicates the possibility of the enhanced risk of joint destruction.

X-rays (Figs 12.10B and C)

In the early stages, diffuse rarefaction is the only finding. Later, other changes develop such as:

- Narrowing of the joint space
- Destruction of the articular surfaces
- Subchondral sclerosis
- Subchondral cysts

Treatment

Medical

Early in the disease, medical line of treatment is as follows:

a. Cold packs to reduce inflammation.
b. Rest to facilitate recovery.
c. Drug therapy.

   In the stage of active inflammation, anti-inflammatory drugs and steroids local as well as systemic are indicated. These are the first line of drugs.

   Disease modifying antirheumatic drugs (DMARD) are the second line of drugs which are given within 3 months of control of acute phase of the disease. Hydroxychloroquine, methotrexate, leflunomide, etanercept, infliximab are some of the common drugs that are used. These drugs act specifically by inhibiting the chemical mediators of inflammation, e.g. TNFα, interleukin-1, etc.

Surgical

Total joint arthroplasty: When considerable joint damage and fibrous/unsound ankylosis has occurred resulting in a painful deranged joint, total joint replacement is the treatment of choice. It should always be done in the remission phase of the disease and never in the active phase of the disease.

Arthrodesis: It is the last option to give a painless, stable joint when all other options have failed. The joint involved is fused in a functional position. More frequently done in wrist and hand.

Reconstructive procedures: These are mainly done for deformities of the hand. Deformities like mallet finger, boutonniere and swan neck are correctable by surgical release and suturing of the slips of the extensor expansion. Once the joints are damaged, such reconstructive procedures are not possible (Ref: For details of the procedures).

Revision Questions

Q. Write notes on:
   a. DMARD
   b. Swan neck deformity
   c. CCP
   d. Pathology of rheumatoid arthritis.

Essay Questions

Q. Discuss the etiopathogenesis, clinical features, radiological features, diagnosis and management of rheumatoid arthritis. Enumerate its complications.

Q. Discuss the drug therapy of rheumatoid arthritis.

HYPERPARATHYROIDISM

Parathyroid glands are responsible for maintaining a fine balance in the levels of calcium in the human body (Fig. 12.11). Normally they are 4 in number, 2 on each side in the vicinity of the thyroid gland. But, the number may vary between 2 and 6. The superior parathyroid glands lie on the posterior surface of the middle 1/3rd of the thyroid and the inferior parathyroid glands lie on the posterior surface of the lower pole of the thyroid.

Classification

The hyperparathyroidism may be classified as:

A. *Primary:* Due to adenoma of the parathyroid.

B. *Secondary:* Due to persistent hypocalcemia, e.g. renal rickets.

C. *Tertiary:* The secondary hyperplasia remains and becomes autonomous.

All types are associated with excessive secretion of parathormone.

Pathology

Excess of parathormone enhances conservation of calcium directly through renal tubular absorption, bone resorption and indirectly through intestinal absorption of calcium by its regulatory action on vitamin D synthesis. Thus, there is hypercalcemia. In

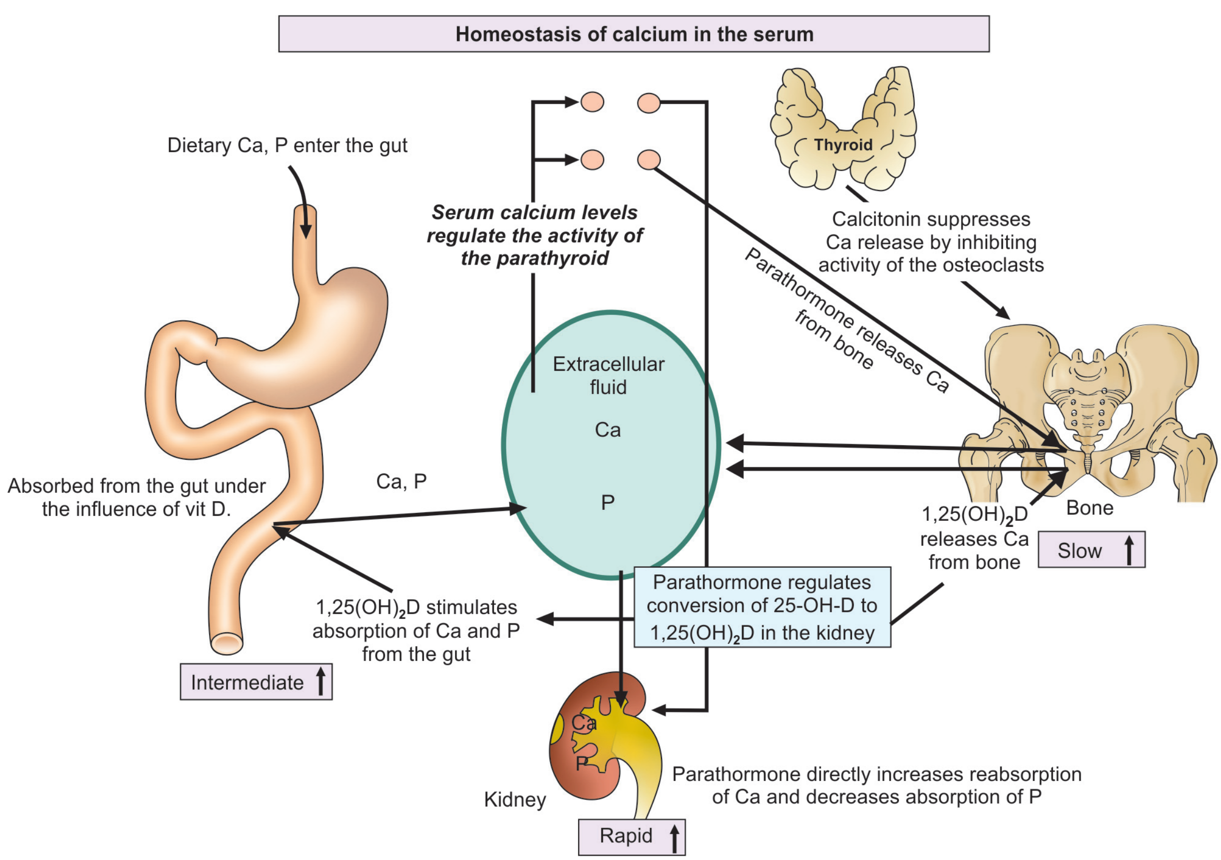


Figure 12.11

Regulation of calcium in the human body. Note that the parathormone has a direct action on the bone and the kidneys and an indirect action on the intestines through the active metabolite of vitamin D. Decrease in serum calcium stimulates the release of parathormone.

spite of augmented renal tubular absorption of calcium, some amount of calcium is excreted in the urine which results in calciuria.

The renal tubular absorption of phosphate is suppressed. Hence, there is hypophosphatemia and phosphaturia.

This abnormality has a generalized effect on the body and the organs.

Effect on Kidneys

It results in calcinosis, nephrolithiasis, recurrent infection and impaired renal function.

Effect on the Bone

It results in loss of bone substance due to direct (resorption) dissolution of bone. Classical subperiosteal erosion, endosteal cavitation and replacement of the marrow by vascular granulation tissue as well as fibrous tissue leads to a classical change known as 'osteitis fibrosa cystica'. Hemorrhage occurring in this fibrous tissue stroma leads to the formation of cysts with fluid which is a brownish in appearance. Hence, these are known as 'Brown tumors'.

The above changes of softening can cause pathological fracture.

Clinical Features

Age: 3rd to 5th decade

Male: Female ratio 1:3.

- Primary hyperparathyroidism may remain asymptomatic. Classical features are remembered as bones, stones, abdominal groans and psychic moans. These are due to hypercalcemia.
- Anorexia, fatigue, weakness, abdominal pain. These depression.
- Nephrocalcinosis and lithiasis.
- Pathological fractures.

Radiological Features

In the Hand

Classical subperiosteal cortical resorption of the middle phalanges.

In the Spine

Biconcave vertebral body due to the bulging of the disk, 'Cod fish spine'.

In the Long Bones

Generalized rarefaction with cystic lesions, osteitis fibrosa cystica and brown tumors.

In the Skull

Stippling calcification. 'Pepper pot skull' (also known as salt pepper appearance).

Laboratory Investigations

- Elevated serum calcium, low serum phosphorus.
- Elevated serum alkaline phosphatase.
- Elevated serum parathormone levels.

Treatment

A. Primary hyperparathyroidism—Is treated nonoperatively with adequate hydration and reduced intake of calcium.

   Indications for surgical removal

   i. Marked and sustained hypercalcemia.
   ii. Progressive nephrocalcinosis and renal calculi.
   iii. Severe bone loss and rarefaction of bone.

   Hungry bone syndrome: This is because of brisk new bone formation that begins to occur after parathyroidectomy. Sudden drop in the levels of serum calcium is observed. This may cause severe tetany. It should be treated by active forms of vitamin D, close monitoring of serum calcium and supplementation when needed.

B. Secondary and tertiary hyperparathyroidism—Treatment should always be directed towards treating the primary pathology.

C. Treatment of pathological/stress fractures—These are always treated by protection and immobilization. Once the primary cause is treated, recalcification begins to occur and the lesions heal.

D. Treatment of deformities—Deformities are always treated by corrective osteotomies after the serum levels of calcium and alkaline phosphatase become normal.

Essay Question

Q. What is the role of parathyroid gland in calcium metabolism? Discuss hyperparathyroidism.

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13 Congenital and Developmental Anomalies

- *Congenital talipes equinovarus*
- *Congenital vertical talus*
- *Developmental dysplasia of the hip (DDH)*
- *Skeletal dysplasia and other conditions*

CONGENITAL TALIPES EQUINOVARUS

It is popularly known by the abbreviation CTEV. It is also known as 'congenital clubfoot'. Hippocrates was the first to describe the condition and mention the importance of gentle manipulation and bandaging for successful correction. In ancient Mexico, the Aztecs treated clubfeet with splints prepared out of cactus and applied casts using flour, lime, tar and cloth. Egyptian tombs have depictions of clubfeet.

Lorenz was the first to advocate the principles of gradual correction. He emphasized the need for correcting adduction deformity first, before correcting varus and equinus. He was also the first to perform Achilles tendon tenotomy.

Since then, several physicians have treated clubfoot successfully using the method of gradual manipulation and splinting. Kites and Ponseti's technique of manipulation, correction and maintenance of correction by the application of serial plaster of Paris casts followed by the use of Denis-Browne splint, are some of the well established nonoperative methods.

Etiology and Pathogenesis

Etiology of clubfoot still remains unknown and is thought to be multifactorial. Following factors are considered:

a. Hereditary
b. Increased intrauterine pressure and malposition (Denis-Browne 1933)
c. Arrest in development
d. Bony abnormalities
e. Tendon and ligament abnormalities

(The clubfoot secondary to neuromuscular disorders are not included in the discussion as they are considered as teratologic).

None of the above mentioned factors have been conclusively proved. But, it is understood that the deformity occurs in the territory supplied by the posterior tibial nerve. Excessive collagen synthesis leads to fibrosis of the muscles and the ligaments resulting in deformity. The deformity is commonly seen in males.

Deformities in Clubfoot

The deformities present are:

a. Equinus
b. Adduction
c. Inversion

These deformities are present both in the forefoot and in the hindfoot.

The varus deformity seen in club foot has both the components of adduction and inversion. All the ligaments of the ankle (except on the lateral side) and tendons of the posteromedial compartment of the ankle are contracted (Figs 13.1A and B). Hence, the deformities are the result of tight ligaments and tendons.

Figures 13.1A and B

Clinical photograph of bilateral clubfoot seen from (A) the front and (B) the back showing all the classical deformities. Note that the deformities are present both in the forefoot and the hindfoot. Also note the size of the heel which is small.

Ligaments Involved

All the ligaments around the talus with the exception of lateral side are contracted.

Medially

i. The deltoid ligament
ii. The talonavicular ligament
iii. The plantar calcaneonavicular ligament (Spring ligament)

Posteriorly

i. Posterior talofibular ligament
ii. Posterior calcaneofibular ligament

Inferiorly

i. The interosseous talocalcaneal ligament.

In addition to this the posterior capsule of the ankle and the subtalar joints along with the plantar fascia are also contracted.

Tendons and Muscles Involved

i. Tibialis posterior
ii. Flexor digitorum longus
iii. Flexor hallucis longus
iv. Tendo-Achilles
v. Abductor hallucis brevis (in the foot)

Diagnosis

It is a straightforward diagnosis. 'CAVE' deformity is seen, i.e. cavus, adductus, varus and equinus. In severe cases, the foot is placed upside down. The size of the heel and the foot is small. Always rule out other associated congenital anomalies and teratological causes, e.g. spina bifida, meningomyelocele, arthrogryposis multiplex congenita, etc. If neglected and the child starts walking with the deformity, callosities start developing on the dorsum of the foot at the site of weight-bearing.

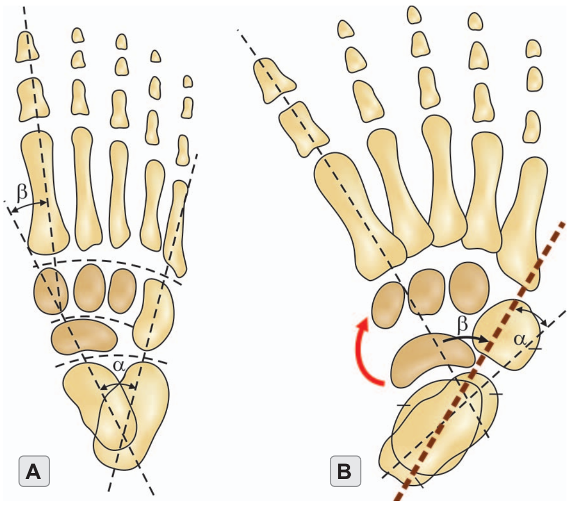
Radiological assessment of clubfoot
(Figs 13.2A and B)

Angles in radiological views	*Normal*	*CTEV*
AP view		
Talocalcaneal angle-α	15–40°	< 15°
Talo-first metatarsal angle-β	0–15°	< 0°
Lateral view		
Talocalcaneal angle	25–45°	< 25°
Tibiocalcaneal angle (Maximum dorsiflexion angle)	40–60°	< 0°

Talocalcaneal Index

The sum of talocalcaneal angle in AP and lateral views gives the index.

40° and more	Excellent
20°–40°	Good
< 20°	Poor



Figures 13.2A and B

Picture showing the angle (α) the talocalcaneal angle in AP view, and (β) the talo 1st metatarsal angle in AP view in (A) normal foot and (B) in clubfoot.

Treatment

Modalities of treatment are classified as nonoperative and operative. The surgical procedures are further classified as:

a. Procedures for early correction.
b. Procedures for correction of residual deformities.
c. Procedures for recurrent clubfoot and for neglected clubfoot.

Age of presentation has an important bearing on the choice of the procedure. However, it is an established fact that an attempted manipulative correction prior to planned surgery definitely lessens the deformity and decreases the extensiveness of the surgical procedure. Hence, treatment should always start with manipulation as the first step.

It is the author's opinion that the manipulation done under anesthesia with relaxed muscles always gives a better correction, especially when the foot is rigid and less flexible.

Nonoperative

Treatment starts at birth. Mother is taught how to manipulate the deformity gently, without inflicting injury, till the child is fit for serial cast application. Milder deformities are managed by corrective strapping and corrective splinting. More severe deformities are managed by serial casts after manipulation based on Ponseti's method (Figs 13.3 and 13.4).

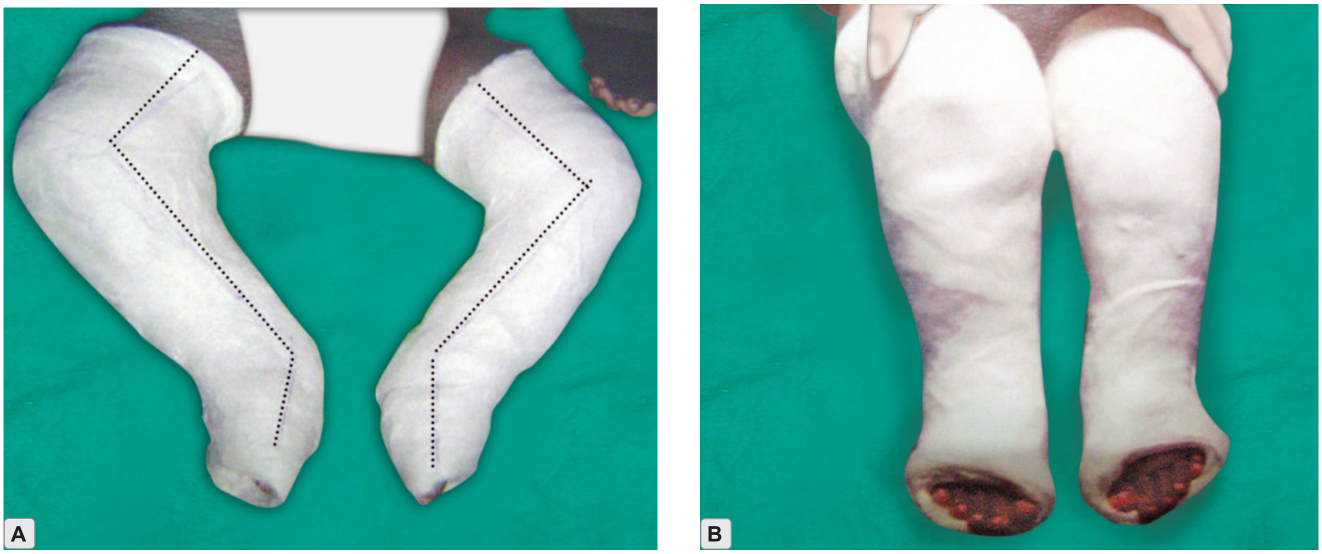
Ponseti's Method of Manipulation (Ignacio Ponseti, 1940)

In this method, the manipulation is done in the following order to correct the deformity. ***First cavus, next adductus, then varus and last equinus (orderly correction of CAVE).*** Non-adherence to these principles may result in poor correction or development of some other deformity, e.g rocker bottom foot, residual forefoot varus (Fig. 13.8).

Figures 13.3A and B

Manipulation being done to achieve gradual correction: (A) Correction of forefoot varus; (B) Correction of hindfoot equinus. Note that the pressure is applied over the sole for correction of equinus and not at the metatarsals.

Note: Without correcting the forefoot varus and achieving some amount of valgus, correction of the hindfoot equinus should never be attempted as it may result in development of rocker bottom deformity.



Figures 13.4A and B

Position of immobilization after (A) full correction of forefoot varus and (B) overcorrected position of the foot after total correction. The cast given should always extend above knee with knee in 90° of flexion to relax the gastrosoleus.

1. *1st, correction of cavus:* This is achieved by supinating and abducting the forefoot which is in contradistinction to other methods which recommend pronation of the first metatarsal for correction of the cavus. The deformity always gets corrected with one manipulation.
2. *2nd and 3rd, corrections of adductus and hindfoot varus:* This is achieved simultaneously by abducting the foot while pressure is applied over talar head. This corrects the major portion of the deformity.
3. *4th, correction of equinus:* Correction should be attempted only when the hindfoot is neutral and the forefoot is in 70° valgus. Foot is always dorsiflexed by applying pressure over the sole of the foot and never under the metatarsal (heel cord tenotomy facilitates the correction of equinus).

Manipulation is done for 1–3 minutes and then the baby is given a feeding bottle while the plaster cast is applied. The cast is changed after 5–7 days and the procedure repeated. Full correction can be achieved with 5–7 casts. A cast given in slightly over corrected position after final manipulation helps in maintenance of correction achieved.

Once the correction is achieved the foot is protected in a splint to prevent recurrence of the deformity. Follow-up is always necessary till 5 years of age (Fig. 13.5). Nonoperative methods can be tried up to 2 years of age.

Figure 13.5

Denis-Browne splint for maintaining the correction achieved. Note how the heel rests on the footplate and is held firmly by the flap shoe with laces thereby maintaining the position of the foot in valgus.

Operative Methods

Surgical procedure should be individualized and limited to the release of tight structures only upto 4 years of age. This is achieved either by dividing them or by lengthening them. Bony correction is indicated in fixed and residual deformities.

For early correction before 12 months. The accepted methods are:

a. Posteromedial soft tissue release, in which the contracted and tight ligaments are released; and the tendons are lengthened by the technique of Z-plasty. (Involved structures that are to be released have been mentioned above). The alignment obtained after the surgery is maintained in a plaster of Paris cast for a period of 6 weeks and then protected with a splint.
b. Peritalar release and stabilization of talonavicular and calcaneocuboid articulation with K-wire. A Cincinnati approach is used for the same. The K-wire is removed at 6 weeks. Postoperative care is the same as above.

(The Cincinnati approach is a transverse approach centered at the level of the tibiotalar joint. It extends from the anteromedial aspect at the naviculocuneiform joint and then over the posterior aspect of the ankle to the anterolateral aspect of the ankle just distal to sinus tarsi) (Fig. 13.6).

For residual deformity after 4 years

a. Hindfoot varus—a calcaneal lateral closed wedge (Fig. 13.7B) or medial open wedge osteotomy (Fig. 13.7A) as suggested by Dwyer.
b. Forefoot varus—a cuboidal close wedge and medial cuneiform open wedge osteotomy is done. Into the open wedge, the wedge of bone taken from the cuboidal closed wedge osteotomy, is inserted (Figs 13.8 and 13.9).

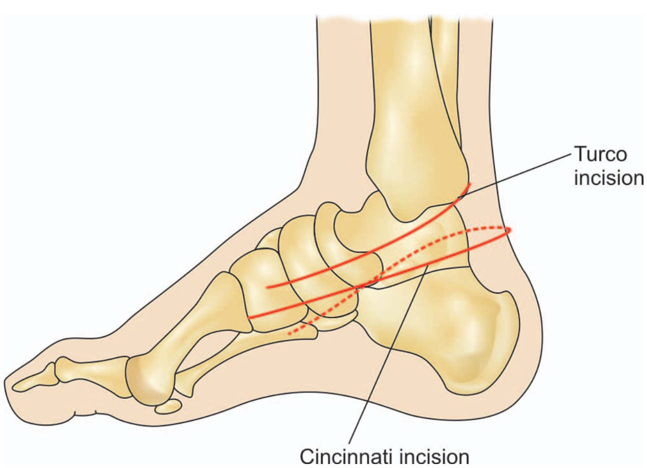
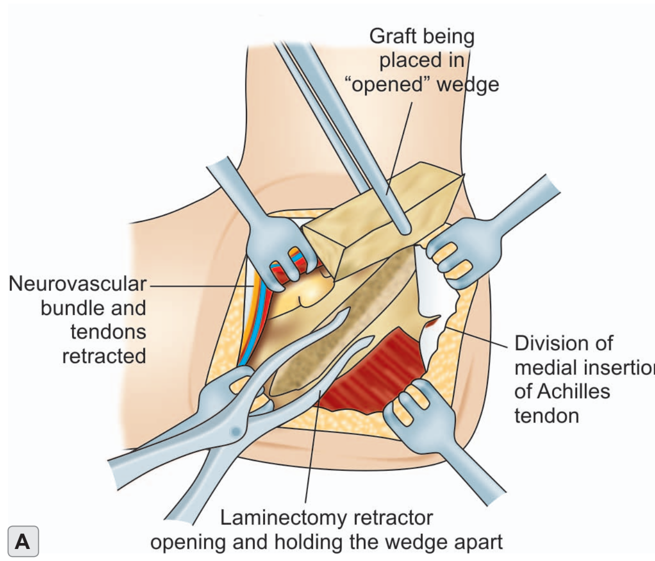
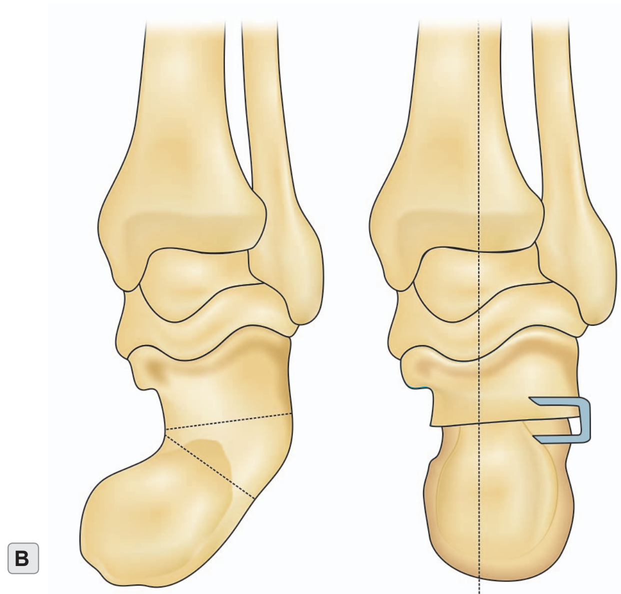


Figure 13.6
Surgical exposures for correction of CTEV.





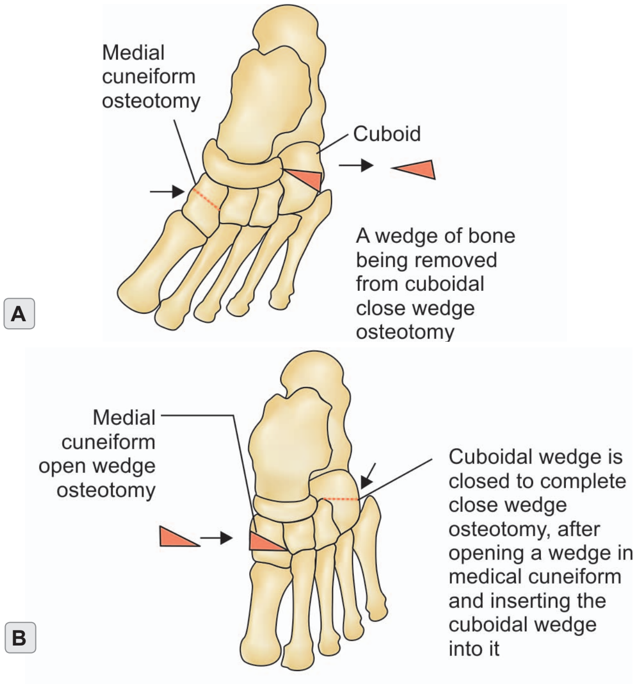
Figures 13.7A and B
Dwyer's osteotomy for the correction of residual heel varus: (A) Medial open wedge; (B) Lateral closed wedge.

For neglected clubfoot and recurrent clubfoot between 4–12 years: **Ilizarov** method or in the younger age group JESS* fixator may be used. These methods not only correct the deformity but also have distinct advantage of lengthening the foot. *Joshi's External Stabilization System.

After 14 years: In skeletally mature foot, wedge osteotomy and triple arthrodesis may be considered. These procedures fuse the talonavicular, calcaneocuboid and subtalar joints (Figs 13.10A to D).

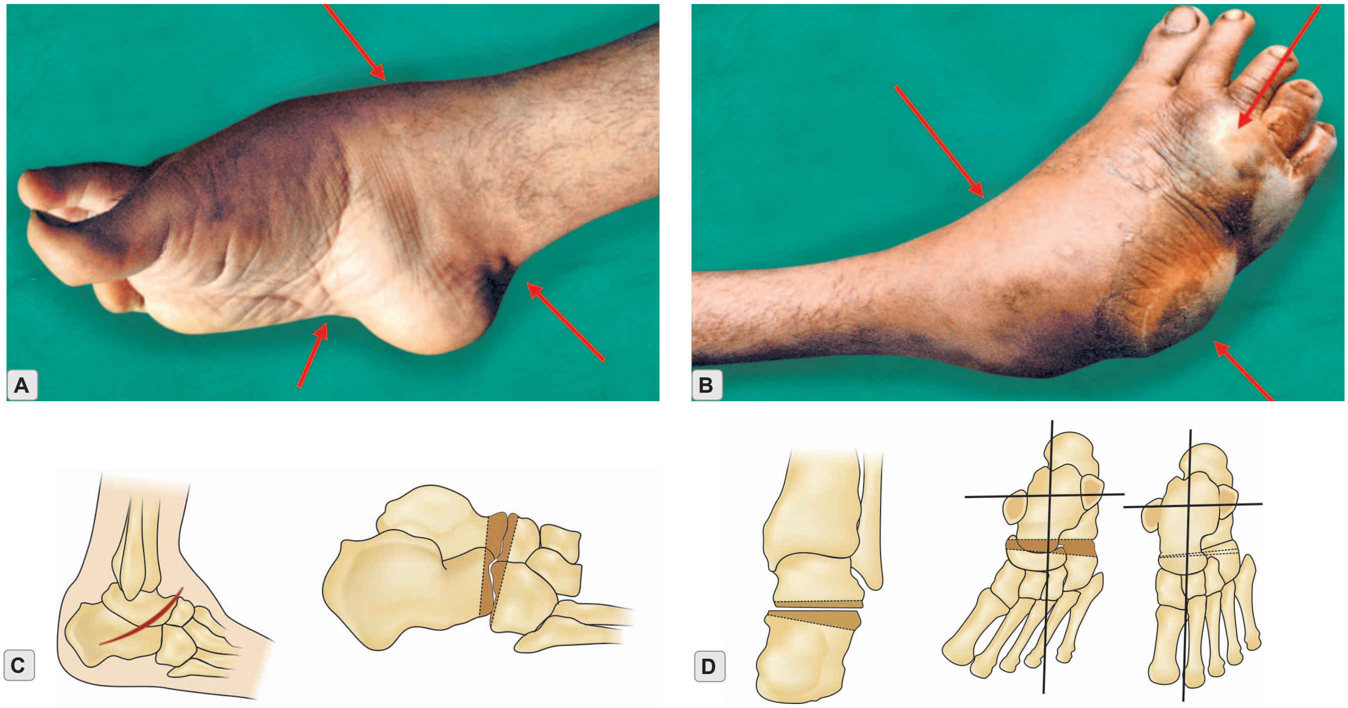
Figure 13.8

Clinical photograph showing residual deformity of forefoot adduction and supination. The child had undergone manipulation and cast application for correction. The residual deformity is perhaps due to the faulty manipulation technique. It is likely that the steps of correction were not carried out methodically (Refer text).



Figures 13.9A and B

Showing the cuboidal close wedge and medial cuneiform open wedge osteotomy for forefoot varus.



Figures 13.10A to D

(A and B) A neglected clubfoot showing a fixed deformity of talipes equinovarus. Note the callosities developed on the pressure points on the dorsolateral aspect of the foot due to weight-bearing stress. One of the callosities is infected. Triple arthrodesis is indicated in such feet for the correction of the deformities. (C and D) The three joints that are fused are, the subtalar, the talonavicular and the calcaneocuboid. Hence, the name triple arthrodesis. The foot should be free of infection before the procedure is done.

Essay Question

Q. Discuss the etiopathogenesis, diagnosis and management of a case of congenital talipes equinovarus. Enumerate the complications and discuss the management.

CONGENITAL VERTICAL TALUS

Heneken was the first one to describe this in 1914.

Synonyms

- Rocker bottom flat foot.
- Congenital convex pes valgus.
- Teratological dislocation of talonavicular joint.

Definition

It is a convex deformity of the plantar surface of the foot occurring due to the vertical disposition of the talus and dorsal displacement of the navicular secondary to dislocation of talonavicular joint.

Etiology

Exact etiology is not known. Following factors may predispose to the occurrence.

a. Muscular imbalance resulting in over action of tibialis anterior.
b. Intrauterine compression.
c. Autosomal dominant transmission.
d. As a part of other associated congenital anomalies, e.g. arthrogryposis, DDH, etc.

Pathological Anatomy

Bone

The talus is fixed in a vertical position with associated hypoplasia of the talar head and the neck. The talonavicular joint is dislocated and the navicular bone is displaced dorsally articulating with the dorsal aspect of the neck of the talus. There is varying degree of subluxation of the calcaneocuboid joint. Thus, there is elongation of the medial column and shortening of the lateral column of the foot.

Ligaments

The tibionavicular and the dorsal talonavicular ligaments are contracted. This prevents the reduction of the talonavicular joint. Also the posterior capsule of the ankle is contracted.

Muscles and Tendons

The tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus brevis and tendo-Achilles are contracted. The tibialis posterior and the peroneal tendons are displaced anteriorly and may act as dorsiflexors instead of plantar flexors.

Diagnosis

The condition is easily diagnosed at birth. The convex plantar surface of the foot, severe dorsiflexion and abduction of the foot, the valgus and equinus position of the heel are the striking features.

Radiological Features (Figs 13.11A and B)

AP view (Fig. 13.12A)

- Increased talocalcaneal angle.
- Forefoot is in abduction.

Lateral view

- Calcaneus is in equinus.
- Talus is vertically placed (normal is horizontal).
- Navicular is displaced dorsally.

Lateral plantar flexion view

- To assess the talar metatarsal and calcaneal metatarsal axis. Both are increased because of equinus position of the bones (Normal values are 3° and 10°, respectively).

Lateral dorsiflexion view

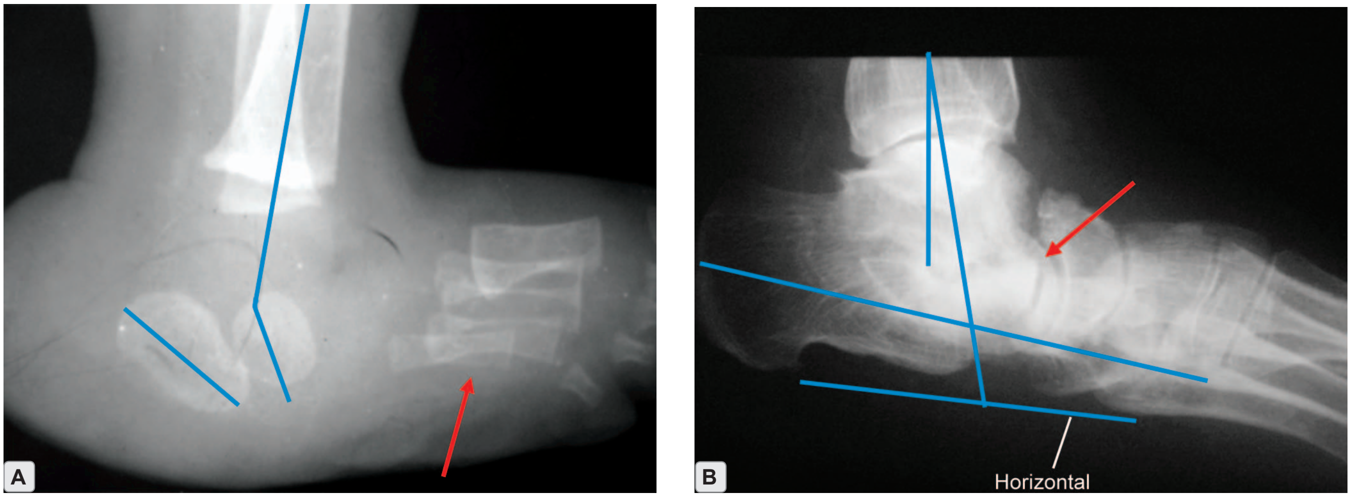
- To assess the heel equinus.

Note: *The lines drawn for assessment of the position of the talus and the calcaneum in relation to the rest of the foot are all drawn along the long axis of the bones. Hence, they derive the names tibiocalcaneal, tibiotalar, talocalcaneal, talohorizontal, talometatarsal, calcaneometatarsal, etc. (Figs 13.11A and B).*

Differential Diagnosis

Congenital Oblique Talus

This resembles congenital vertical talus but with certain differences as shown in Figures 13.12A to C.



Figures 13.11A and B

(A) An infant with congenital vertical talus. (B) An adult with neglected congenital vertical talus with secondary degenerative arthritis. (A) The soft tissue shadow shows the convexity of the plantar surface of the foot. The talus is near vertical and the calcaneum is in plantar flexion. The navicular bone has not appeared but note the position of the metatarsals. They are dorsally displaced indicating the dorsal displacement of the navicular. (B) An adult with rocker bottom foot. Note that the tibiotalar and the talohorizontal angle are near 180° and 90°, respectively.

Figures 13.12A to C

(A) Congenital oblique talus showing increased talocalcaneal angle in AP view. Note that it has all the features of a vertical talus except that; (B) The talonavicular joint subluxates in neutral position of the foot; and (C) Gets reduced in equinus. The red arrow shows the same.

Treatment

a. Nonoperative treatment starts at birth. Manipulation of the foot into plantar flexion and inversion in an attempt to reduce the dislocation is carried out. Rarely successful and if successful the reduction is maintained with closed pinning and subsequently removal of pin and application of splints is done. Even if it is not successful, it helps in future surgery by stretching the soft tissues.
b. Surgical treatment consists of open reduction of dislocation and maintaining the correction achieved. This is done in single stage or in multiple stages. Ideal time for surgery is at 1 year. More severe is the deformity more extensive is the surgery.

One Stage Release

1st step: Reduction of talonavicular joint by release of tibialis anterior, tibionavicular, talonavicular ligaments and the capsule. Reduction is stabilized with a pin.

2nd step: Lengthening of peroneals and toe extensors along with reduction of calcaneocuboid joint.

3rd step: Correction of equinus contracture, release of Achilles tendon and capsule of the subtalar and the ankle joints.

Figure 13.13

Neglected Rocker bottom foot in a middle aged person. Note the degenerative changes in the subtalar, talonavicular, and the calcaneocuboid joint. Patient was treated by triple arthrodesis.

Appropriate cast is given postoperatively which is changed periodically and maintained for at least 8–12 weeks.

Excision of the navicular bone with Grice Green arthrodesis is indicated in older children and in resistant deformities.

Always keep a watch on recurrence during the follow-up, up to 8–10 years of age.

Complication of a neglected rocker bottom foot is tarsal degeneration resulting in degenerative arthritis. This occurs because of altered biomechanics during stress loading (Fig. 13.13). It is treated by triple arthrodesis.

Essay Question

Discuss the pathomechanics, diagnosis and management of rocker bottom flat foot.

DEVELOPMENTAL DYSPLASIA OF THE HIP (DDH)

The abbreviation CDH (congenital dislocation of the hip) is now replaced by DDH (developmental dysplasia of the hip). DDH is more appropriate because there is no congenital embryonic anomaly as such. The development starts normally in an embryo and due to some extraneous factors the head of the femur is not contained in the acetabulum. The most common factor is the laxity of the capsule. Other factors like malposition of the femoral head and the effect of abnormal stresses on the developing femoral head also play a part in the pathology. The child may be born with dislocated/subluxated hip or born with dislocatable and relocatable/reducible hip. In either case, the hip is reducible with manipulation. According to Klisic (1989) “developmental displacement of the hip is a dynamic disorder potentially capable of becoming better or worse as the child develops”. This statement is absolutely true. If the hip remains dislocated for longer periods structural changes develop and reduction becomes progressively difficult. But, if the hip is maintained in a position of reduction right from infancy, it becomes normal with growth. So, the statement supports the fact that the posture has a great role to play in the origin, progress and the regress of the pathology (Fig. 13.14).

Thus, DDH is not only multifactorial in origin but also may present differently in different age groups.

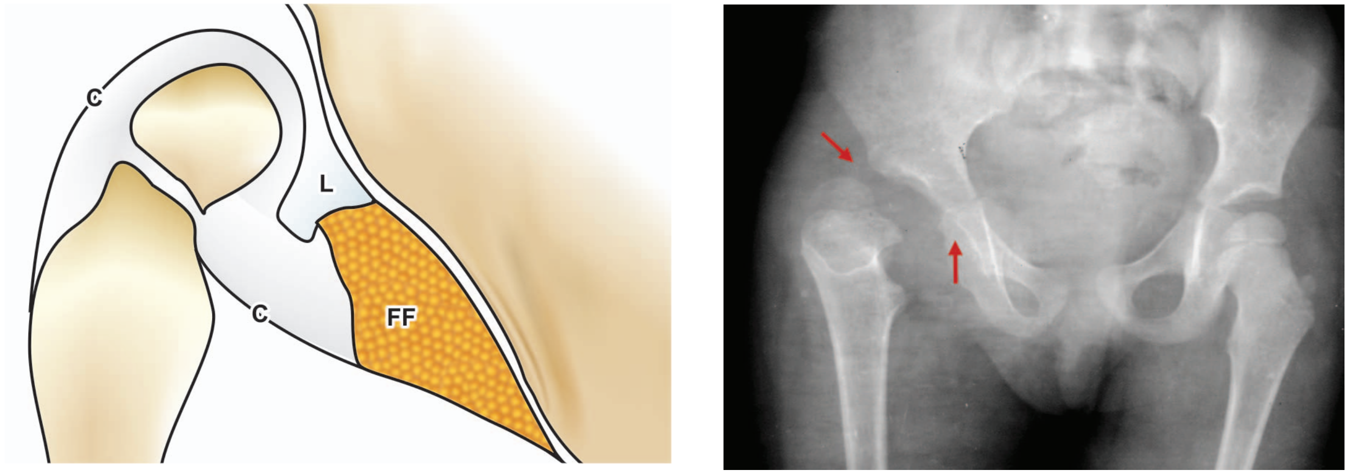


Figure 13.14

Diagrammatic representation and the radiograph of a neglected DDH. FF—shows the acetabulum filled with fibrofatty tissue, L—the limbus is inverted and C—the capsule is stretched.

Predisposing Factors

a. *Racial dominance:* Commonly seen in Native Americans and Caucasians. Not common in Asians and Africans.
b. *Ligamentous laxity:* Studies by Wynne-Davis suggests that there is a genetic predisposition for the ligamentous laxity. This is the most common pathology that causes the hip to dislocate. However, this does not explain unilateral hip dislocation. Also suggests the postural influence on the development of DDH.
c. *Prenatal positioning:* A frank breech presentation has a higher incidence when compared to footling breech.
d. *Postnatal positioning:* Incidence has been found high in Native Americans who carry their babies wrapped up with the hip in full extension in cradle board.

   Considering these facts, it can be concluded that relatively gentle factors applied persistently cause deformation and dislocation/subluxation.
e. Primary acetabular dysplasias.
f. *Other associated anomalies:* DDH may be a part of other associated congenital anomalies, e.g. torticollis, congenital dislocation of the knee, etc.
g. *Sex:* The incidence of DDH is more in females when compared to males.

Clinical Features

Up to 2–3 Months (the neonate)

The hip is always dislocatable and reducible. The classical tests of Barlow's and Ortolani's are always positive.

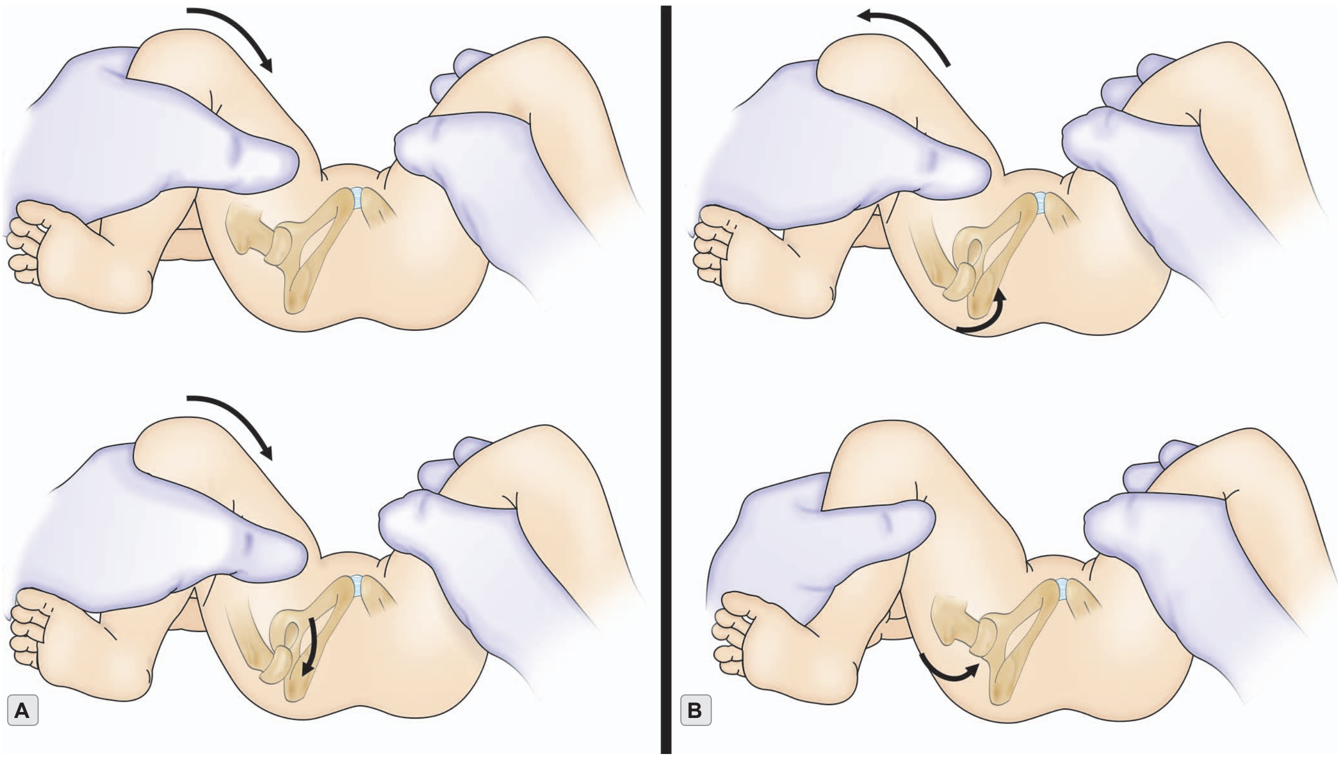
Barlow's Test (Fig. 13.15A)

With the baby in supine position (on the couch or mother's lap) the examiner holds baby's both the knees and gives a gentle adduction push to one of the knees. If the hip is dislocatable, the feel of femoral head jumping out of the acetabulum is made out by the fingers placed in the region of the greater trochanter.

The release of pressure slips the head back into the acetabulum.

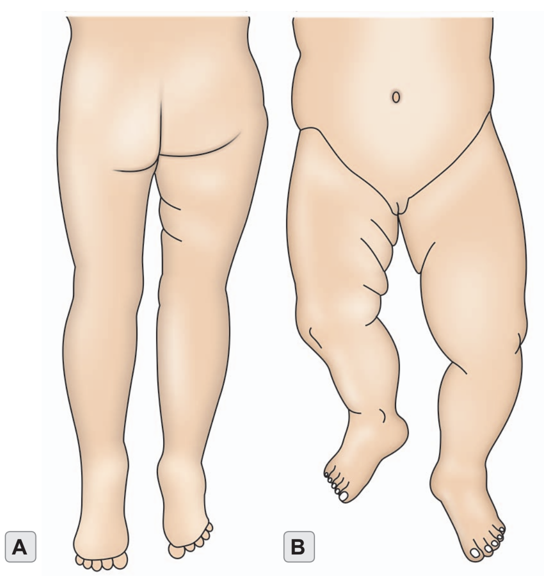
Ortolani's Test (Fig. 13.15B)

This is the reverse of Barlows test. In this test, the examiner tries to reduce the dislocated hip. The test is performed with the baby in supine position as in Barlows test. The babies thigh is grasped between the thumb and the index finger. With the other fingers, the greater trochanter is gently lifted up, abducting the hip at the same time. The clunk of reduction is felt. When the hip is adducted, the head gently slips out (Barlow's test).



Figures 13.15A and B

Picture showing tests of (A) Barlow's and (B) Ortolani's being performed to dislocate and relocate/reduce the hip.



Figures 13.16A and B

Asymmetry of skin creases on the involved side in a unilateral DDH.

These tests have to be repeated a couple of times to make certain that there is DDH. They should also be performed on the other hip to assess the status of the other hip.

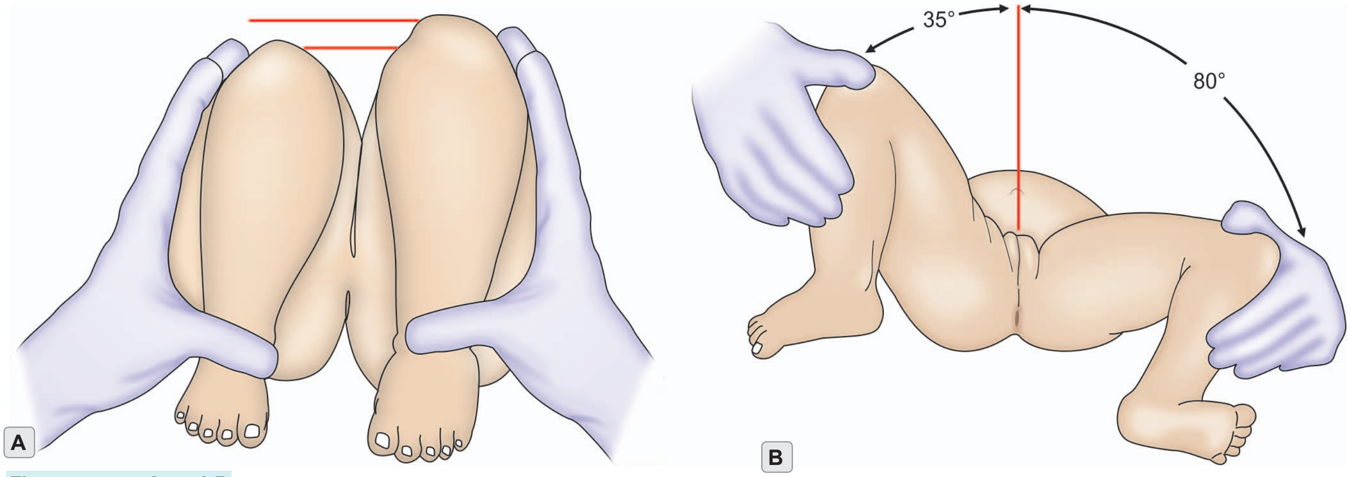
After 3–6 Months (the infant)

During this period the irreducibility becomes gradually established. As the hip remains in dislocated position, other physical findings appear.

a. Asymmetry of the thigh and gluteal folds (Figs 13.16A and B).
b. Shortening of the thigh.
c. Superior location of the greater trochanter.
d. Positive telescopy.
e. Limitation of abduction (Fig. 13.17B).
f. Discrepancy in the levels of the knee (Allis', Perkins or Galeazzi sign) (Fig. 13.17A).
g. Klisic test (Figs 13.18A and B).
h. Vascular sign of Narath may be positive and femoral artery pulsation may be less well felt.

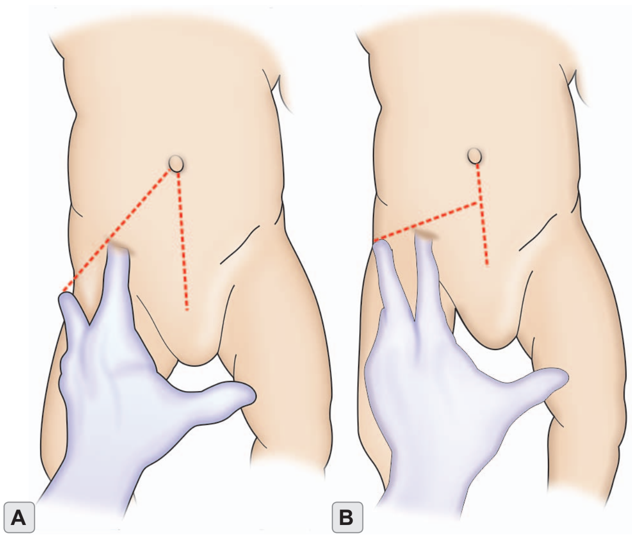
Klisic test: With the child supine the index finger is kept over the anterior superior iliac spine and the middle finger over the greater trochanter. Next, an imaginary line is drawn between the two. This line when extended upwards cuts through the umbilicus in a normal hip.

In DDH, because the trochanter is elevated this line passes half way between the umbilicus and pubis.



Figures 13.17A and B

(A) Asymmetry in the level of the knees which is known as Allis', Perkins or Galeazzi sign. On the involved side in a unilateral DDH, the knee will be at a lower level; (B) Limitation of abduction of the involved right hip.



Figures 13.18A and B

Klisic test. Note the imaginary line cutting through the umbilicus in a normal hip and cutting midway between the umbilicus and the pubis in a DDH.

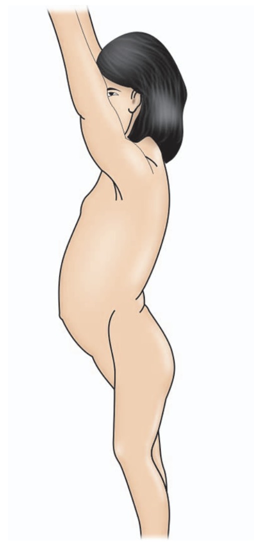


Figure 13.19

Exaggerated lumbar lordosis indicating the presence of flexion deformity in a neglected, bilateral DDH. The child will have a waddling gait.

After the Child Begins to Walk

a. The shortening becomes pronounced in unilateral cases.
b. Trendelenburg gait develops in unilateral cases and waddling gait in bilateral cases.
c. Exaggerated lumbar lordosis secondary to flexion contracture at the hip becomes noticeable (Fig. 13.19).

Note: *Delay in milestones may or may not be observed. However, if there is a delay in walking, the possibility of DDH should be kept in mind.*

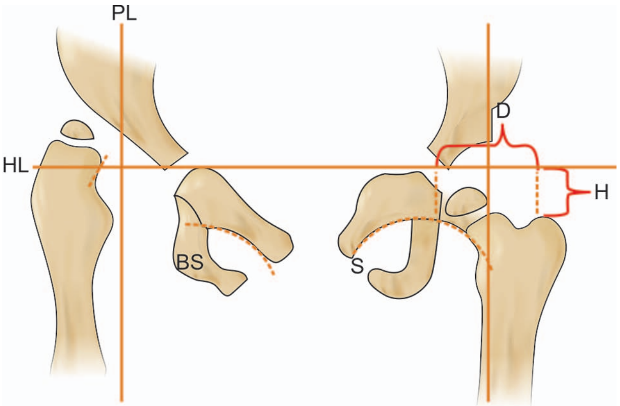


Figure 13.20

Picutre showing the following:

HL—Hilgenreiner's line. It is a horizontal line drawn passing through the center of the triradiate cartilage.

PL—Perkin's line. It is a line drawn perpendicularly down from the bony edge of the acetabulum.

S—Shenton's line. It is a line along the neck of the femur to the inner margin of the pubic bone. It forms a smooth 'C' shaped curve.

BS—Broken Shenton's line. This is seen in subluxation/dislocation

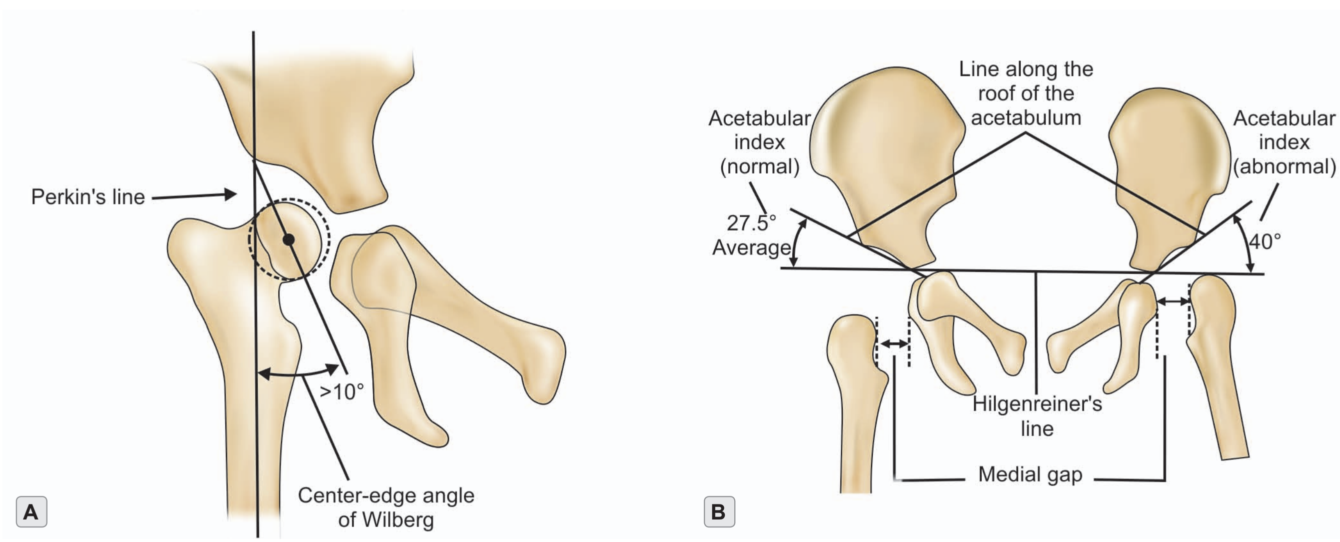
H—Dimension H. This is the height measured from the top of the ossified femur to the Hilgenreiner's line.

D—Dimension D. This is distance measured from the inner border of the pelvic tear drop to the center of the tip of the ossified femur.

Dimension H and D quantify proximal and lateral displacement of the hip respectively. They are useful even at an age where the epiphysis of the head of the femur is not ossified.

Investigations

a. Ultrasound is a useful investigation in the newborn in whom X-ray imaging is of no use because of cartilaginous nature of the bones. It should be used judiciously and findings should be always be correlated with clinical findings to prevent overdiagnosis (for more information refer α and β angle of Graf).
b. Magnetic resonance imaging (MRI) is a better investigation than ultrasound. But, it is expensive and is accompanied by the disadvantage of sedating the newborn.
c. X-ray gives useful information in an infant, only after 3 months.
   With the help of Hilgenreiner's, Perkin's and the Shenton's line the location of the femoral head can be assessed. Also, the center edge angle of Wilberg and the Acetabular index can be measured (Figs 13.20 and 13.21).
   Hilgenreiner's and the Perkin's line divides the hip into four quadrants. In a normal hip, the epiphysis of the femoral head lies usually in the lower inner quadrant. A subluxated or dislocated hip the epiphysis of the femoral head lies in/close to the upper outer quadrant.
d. Arthrography gives useful information regarding the concentric nature of reduction.



Figures 13.21A and B

(A) *Center-edge angle of Wilberg:* It is an angle formed between the Perkin's line and the line drawn from the lateral lip of the acetabulum passing through the center of the femoral head. In older children (10–13 years) the angle should always be more than 10°; (B) *Acetabular index:* This is an angle formed between a line drawn along the margin of the roof of the acetabulum and Hilgenreiner's line average angle in newborn is about 27.5°. It decreases with age. *Medial gap:* This is the distance between the inner margin of the tear drop and the inner margin of the neck of the femur. The gap increases in dislocation. Always compared with the opposite hip. Not useful in bilateral cases.

Treatment

Nonoperative

Nonoperative treatment is indicated in neonates, infants and young children with reducible hips. Treatment starts soon after birth. It aims at maintaining the femoral head in the acetabulum till the relation gets organized and the risk of redislocation gets eliminated.

The reducible hips are *immobilized in reduced position* with the help of splints and/or plaster of Paris casts. Following this, they are *watched* carefully *for* the possibility of *redislocation, avascular necrosis and nerve palsies,* e.g. femoral nerve in Pavlik harness (Figs 13.22 and 13.23).

Open Reduction

Surgery of open reduction is done in older children (6 months–6 years). In the young (<6 months), open reduction is done only when closed reduction fails. Those hips which remain in dislocated position for a considerable time, are not amenable for closed methods of reduction. The adaptive changes which take place in the soft tissues and later in the bone prevent reduction. Preoperative traction in the desirable position has proved its usefulness beyond doubt. It should always be given before open reduction. This is known to reduce the risk of avascular necrosis (AVN)(Fig. 13.24).

When structural skeletal changes have not taken place release of constricted/contracted soft tissues such as muscles and tendons, capsule and ligaments, excision of the limbus, etc. will help in reducing the head into the acetabulum.

When structural skeletal changes have taken place, femoral osteotomy and femoral shortening procedures for containment OR shelf operations and osteotomies of the ilium for deepening the shallow acetabulum, are indicated. These procedures help in containing the head of the femur in the acetabulum (Fig. 13.25).

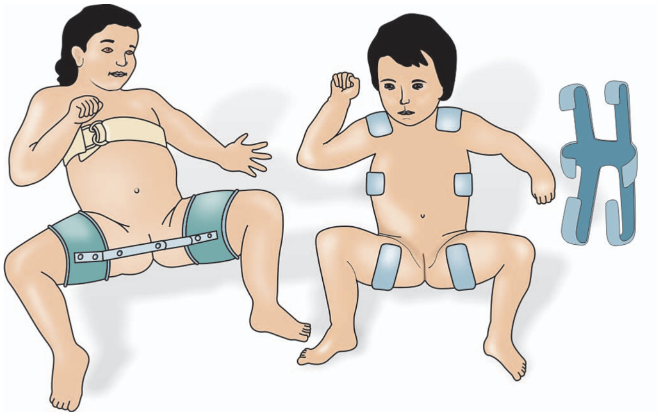


Figure 13.22

Craig splint and Von Rosen splint.

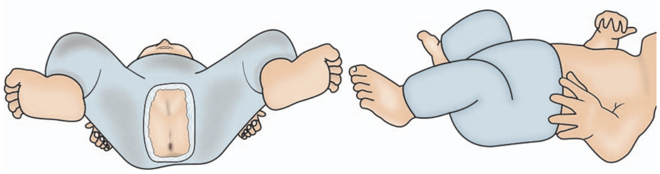


Figure 13.23

Position of immobilization in spica cast after closed manipulation and reduction.

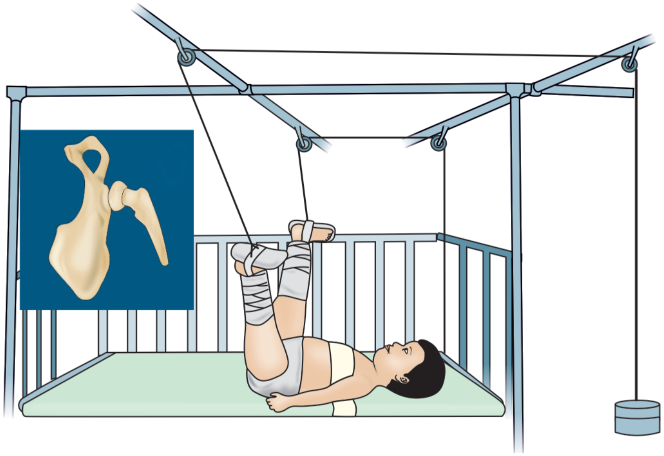


Figure 13.24

Bryant's traction/Gallows traction.

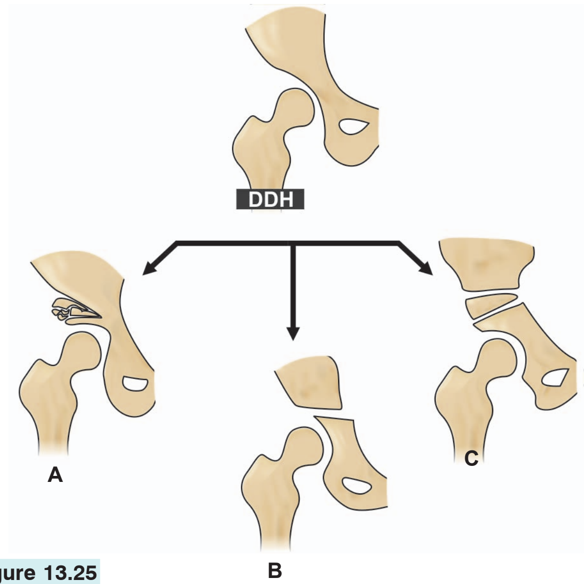


Figure 13.25

Different types of containment procedures for DDH. A—Shelf procedure; B—Chiari's osteotomy; C—Salter's osteotomy.

Reconstructive Procedures

These procedures are considered is older children and adults where containment is not possible because of long-standing pathology.

In adults with unilateral dislocated hip, *limb lengthening procedures* are considered. These procedures take care of the shortening and the limp. In those hips which become painful (AVN), *total hip replacement* may be considered.

Revision Questions

Q. Write notes on:
- a. DDH
- b. Barlow's test
- c. Ortolani's test.
- d. Hilgenreiner's and Perkin's line.
- e. CE angle of Wilberg.
- f. Salter's innominate osteotomy.
- g. Galeazzi sign

Essay Question

Q. Discuss the etiopathogenesis, diagnosis and management of a case of DDH. Enumerate the complications and discuss the management of complications.

SKELETAL DYSPLASIA AND OTHER CONDITIONS

Skeletal Dysplasias

Skeletal dysplasias are a group of disorders that result in disturbances in the normal growth of the skeleton. They are genetically determined. Most of them have an autosomal dominant type of inheritance. Few of them are autosomal recessive OR X-linked.

The terminologies and the pattern of dysplasias have been clearly explained by Rubin P through his dynamic classification.

Dynamic Classification of Bone Dysplasias

(Based on Rubin P Dynamic Classification of Bone Dysplasias. Chicago year book medical publishers, 1964, p 82). According to this dynamic classification, the dysplasia is the result of either hypoplasia or hyperplasia of the specific cells in different regions of the bone, i.e. epiphysis, physis, metaphysis and diaphysis. Hypoplasia is deficient or failure and hyperplasia is stimulation and excess.

1. Epiphyseal dysplasias

A. Epiphyseal hypoplasias

- i. Articular cartilage, e.g. spondylo-epiphyseal dysplasia congenita and tarda.
- ii. Ossification center, e.g. multiple epiphyseal dysplasia congenita and tarda.

B. Epiphyseal hyperplasias

- i. Articular cartilage, e.g. dysplasia epiphysealis hemimelica

2. Physeal dysplasias

A. Cartilage hypoplasias

- i. Proliferating cartilage, e.g. achondroplasia congenita and tarda.
- ii. Hypertrophic cartilage, e.g. metaphyseal dysostosis congenita and tarda.

B. Cartilage hyperplasias

- i. Proliferating cartilage, e.g. hyperchondroplasia.
- ii. Hypertrophic cartilage, e.g. enchondromatosis.

3. Metaphyseal dysplasias

A. Metaphyseal hypoplasias

- i. In formation of primary spongiosa, e.g. hypophosphatasia.
- ii. In absorption of primary spongiosa, e.g. osteopetrosis congenita and tarda.
- iii. In absorption of secondary spongiosa, e.g. craniometaphyseal dysplasia congenita and tarda.

B. Metaphyseal hyperplasias

- i. Spongiosa, e.g. multiple exostosis.

4. Diaphyseal dysplasias

A. Diaphyseal hypoplasias

- i. Periosteal bone formation, e.g. osteogenesis imperfecta congenita and tarda.
- ii. Endosteal bone formation, e.g. idiopathic osteoporosis congenita and tarda.

B. Diaphyseal hyperplasias

- i. Periosteal bone formation, e.g. progressive diaphyseal dysplasia.
- ii. Endosteal bone formation, e.g. hyperphosphatasemia.

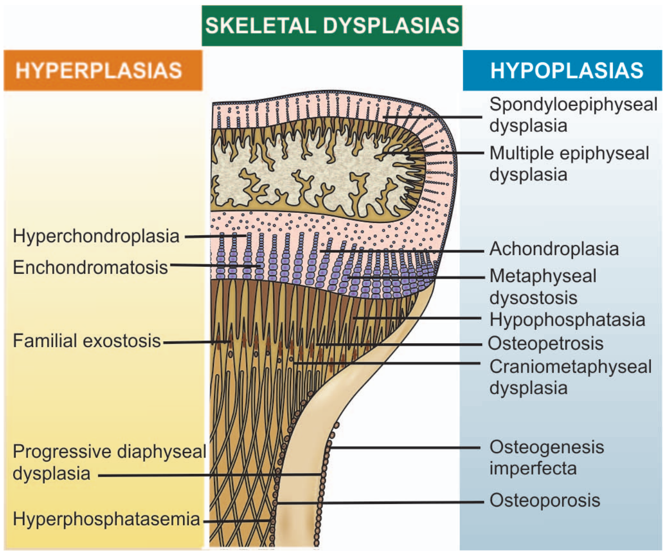


Figure 13.26

Areas of bone responsible for skeletal abnormality and different dysplasias.

IMPORTANT SKELETAL DYSPLASIAS (FIG. 13.26)

Spondyloepiphyseal Dysplasia

This is characterized by generalized involvement of the epiphysis of the spine and long bones resulting in disproportionate dwarfism.

Types

a. Congenita—severe form presents at birth. Disproportionate dwarfism is pronounced.
b. Tarda—less severe form presents in early childhood with less disturbance in growth. Loss of height is minimal.

Transmission: Autosomal dominant.

Clinical features

- Wide set eyes
- Short neck
- Barrel chest
- Exaggerated lumbar lordosis with protuberant abdomen
- Coxa vara with waddling gait
- Angular deformity in the lower limb, e.g genu valgum.

Radiological features

- Delay in the appearance of the epiphysis (is responsible for the short stature)
- Coxa vara in the hip
- Genu valgum in the knee
- Platyspondyly in the spine
- Hypoplasia of the odontoid process.

Orthopedic care

The care is directed towards *correction of angular limb deformities.* This possibly prevents the development of degenerative arthritis at a later period.

Odontoid hypoplasia may result in *atlantoaxial instability.* When present it needs to be *stabilized.*

Multiple Epiphyseal Dysplasia

The condition was first described by Fairbank as Dysplasia Epiphysealis Multiplex.

In this unlike spondyloepiphyseal dysplasia, the spine, skull and vertebra are not involved. The epiphysis of the long bones and short tubular bones are involved. Transmission is autosomal dominant; rarely recessive. Delay in appearance of epiphyseal ossification centers is observed radiologically mainly in long bones and short tubular bones. When it appears, the epiphysis is small, flat and may have mottled appearance. Early diagnosis and correction of the deformity is indicated to prevent early degenerative arthritis.

Achondroplasia

It is the most common form of dwarfism. The incidence is around 1.5–2.0 per 10,000 live births (Fig. 13.27).

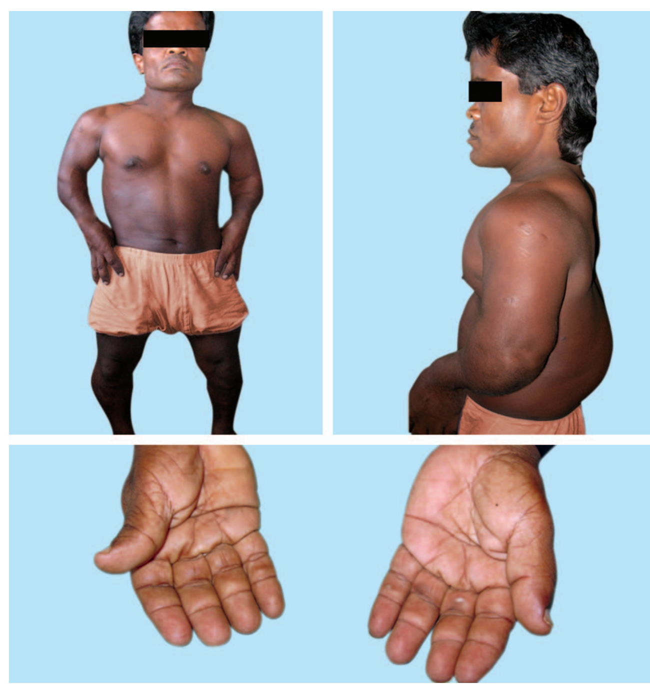


Figure 13.27

Clinical features of an achondroplastic dwarf. Note the 'Starfish hand' on one side (Right hand) and 'Trident hand' on the other side (Left hand). Also note the presence of a single transverse crease in the palm.

Types

- *Spontaneous mutation occurring in paternal gene* (more common than maternal) of normal parents. The *offspring* is an *achondroplastic dwarf.* Can expect *normal life expectancy.*
- *Homozygous achondroplasia* occurring in the *offspring of achondroplastic parents*. Generally the *outcome is fatal* in the neonatal period.

Transmission: Autosomal dominant with complete penetrance.

Clinical features (Fig. 13.27)

- Normal intelligence.
- Bossing of the skull.
- Small maxillae with prominent mandibles.
- Deformities in the spine.
- Starfish Hand: Short stubby hand with shortening of the middle finger resulting in equal length of all the digits.
- Trident Hand: The middle and the ring fingers are separated by a greater space.
- Short upper and lower limbs.
- Protuberant abdomen.

Radiological features

- Shortening of the tubular bones with increased diameter.
- V or U shaped growth plate of the distal femur.
- Widening of the metaphysis of long bone.
- Squared ilium with horizontal acetabulum.
- Posterior scalloping of the vertebral body.
- Narrow spinal canal with decreased interpedicular distance.
- Shortness of the base of the skull with small foramen magnum.

Orthopedic care

Care is necessary for cervical spinal cord compression in young infants and lumbar cord compression due to canal stenosis in older age group. The treatment is decompression of the foramen magnum/spinal canal.

Short limbs may need limb lengthening procedures. Lengthening of limbs involves both limbs and procedure extends over a very long period of time. Hence,it is to be considered carefully.

Hypochondroplasia

This is a less severe form of dwarfism resembling achondroplasia. Transmission is autosomal dominant. The condition cannot be diagnosed at birth but presents itself at a later period. Limb lengthening procedures are indicated to correct short stature.

Osteopetrosis

This is also known as Albers-Schönberg disease (after Albers-Schönberg who described the disease in the year 1904), Marble bone disease and chalk bone disease. Failure of osteoclastic resorption leads to the formation of calcified cartilage and primitive osteoid. Thus, there is increased sclerosis and brittleness of bone with a tendency for easy fracture.

Histopathology identifies abundant osteoclasts. But these osteoclasts do not respond to Parathormone (PTH) because of inherent abnormality. No activation of macrophages and monocytes is observed. Thus, calcified cartilage and primary woven bone is found in abundance as it not replaced by lamellar bone. This immature bone is tightly embedded into the coarse fibrous matrix. This increases the fragility of the bone and makes it susceptible for pathological fracture.

Types

- *Malignant form* in which there is *obliteration of the marrow* resulting in *pancytopenia, anemia,* hepatosplenomegaly, easy bruising, bleeding abnormalities and delayed dentition and caries teeth. Bony overgrowth leads to *obliteration of the cranial foramina* leading to *nerve palsies.* Blindness and deafness may develop. *Pathological fractures* are common. The condition is *lethal.* Bone marrow transplantation is the only hope.
- *Benign or tarda form* in which the diagnosis is incidental. No clinical signs are present. Patients have a normal life span. Pathological fracture and premature osteoarthritis can occur.
- *Associated with renal tubular acidosis.* In this form, the lack of carbonic anhydrase interferes with the acidification of the bony surface and interferes with the resorption by osteoclasts. Except anemia, other features are same as that of malignant form.

Inheritance

- Malignant form is transmitted as autosomal recessive.
- Benign or tarda form is transmitted as autosomal dominant.
- Tubular acidosis form is transmitted as autosomal recessive.

Clinical features

- Features of malignant form (see above).
- Pathological fractures and deformities.
- Bone pain and back pain.
- Premature osteoarthritis.
- Osteomyelitis (mandible).

Radiological features

- Increased density of bone.
- Filling of intramedullary canal with bone.
- Endo bone formation (bone within the bone).
- 'Rugger jersey' spine (as a result of sclerosis near the end plates of the vertebra)
- Dense skull bone with shallow fossae.

Orthopedic care

- Malignant form is treated by early bone marrow transplantation.
- Benign or tarda form does not require any active treatment unless pathological fracture occurs.
- Associated acidosis is treated by alkaline therapy.
- Pathological fractures in the pediatric age group are treated by closed reduction and cast application. Healing occurs eventually.
- Adult fractures need reduction and internal fixation. The procedure is technically highly demanding because drilling and reaming is extremely difficult in these bones.
- Joint replacement is done for premature degenerative arthritis.
- Complication of cranial nerve impingement is treated by foraminotomy and decompression
- Anemia and thrombocytopenia is treated by administration of erythropoietin.
- Interferon-gamma is used to allow bone resorption by enhancing superoxide production.
- Calcitriol in high doses can be given in an attempt to promote osteoclastic resorption.
- Medical treatment improves the condition but does not cure it.

Paget's Disease

Described by Sir James Paget, a British surgeon in the year 1877.

It is a disease with an imbalance between normal osteoclastic and osteoblastic activity of a bone resulting weakening, deformities and pathological fractures. Hence, it is also known as 'osteitis deformans'.

Etiology

a. *Viral*: Some of the viruses associated are paramyxovirus, canine distemper virus, etc.
b. *Genetic:* Paget's disease is seen among siblings, which suggests the presence of a hereditary factor. Some genes have been identified, e.g. mutation of PDB2 on chromosome 18q22.1 which encodes RANK protein.

Pathology

Bones affected are that of the spine, pelvis, skull, femur, and tibia, fibula.

Generally, involvement is polyostotic. Very rarely, it can be mono-ostotic. The basic pathology is that of a massive turnover of bone with increased osteoclastic activity to begin with followed by increased osteoblastic activity.

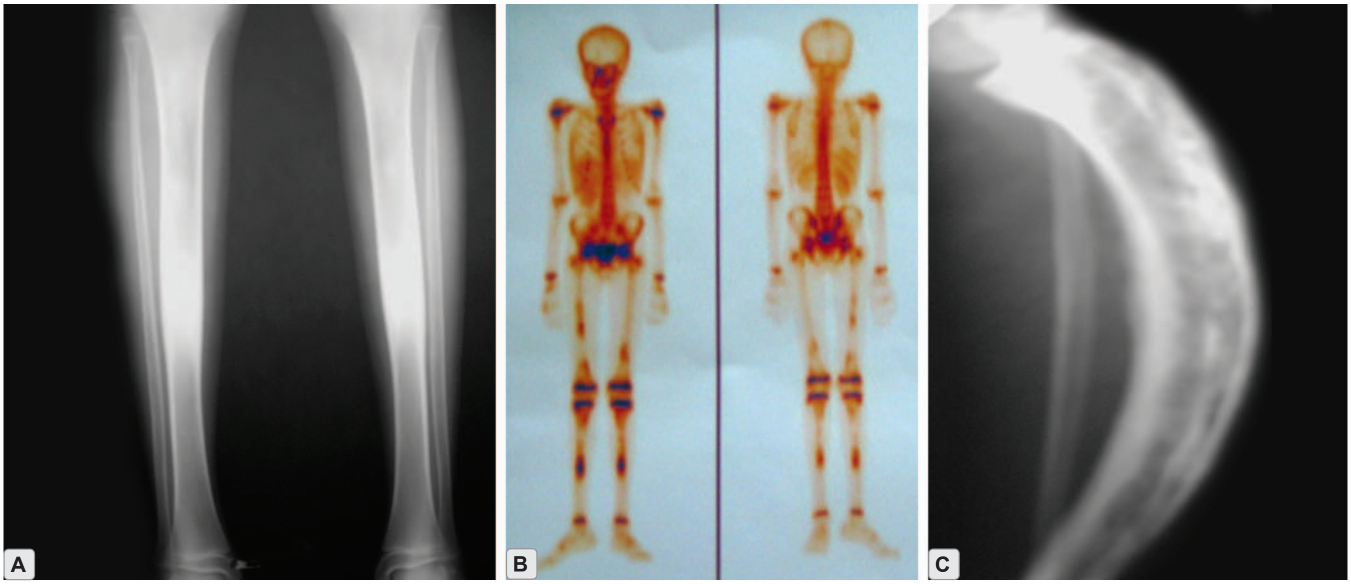
Three distinct stages are recognized:

i. *Osteolytic or hypervascular phase:* In this phase, there is increased osteoclastic activity followed by an intense osteoblastic activity which ultimately results in disorganized mass of primary woven bone formation in the areas affected. The blood flow increases to such an extent as to form AV shunts in the bone there by causing significant increase in cardiac output and strain on the heart.
ii. *Intermediate phase:* In this phase, there is mixed activity, predominantly osteoblastic. Deformities begin to develop in this phase of the disease.
iii. *Burnt-out or quiescent phase:* The exaggerated activity stops. The vascular fibrous tissue replaces the marrow.

   Absence of haversian system and development of sclerotic bone is characteristic of Paget's disease. Remodeling is poor and the quality of bone changes.

Clinical signs and symptoms

- Bone pain is a common feature.
- Constitutional symptoms like headache and drowsiness.
- Neuritic pains along the nerves, e.g. in skull here the nerves get compressed in the foramina.
- Arthritis.
- Deformities in long bones.
- Pathological fractures.



Figures 13.28A to C

(A) Symmetrical hyperdense areas in the tibia suggestive of Paget's disease; (B) The bone scan shows increased uptake of the neucleotide; (C) The characteristic bowing of both anterior and posterior cortices of the tibia seen in a mono-ostotic Paget's disease.

Investigations

- *Laboratory*
  i. Increased serum alkaline phosphatase with normal serum calcium and phosphorus.
  ii. Increased serum and urinary hydroxyproline which is an indicator of bone lysis.
- *Radiology (Figs 13.28A and C)*
  i. The involved bones show areas of lysis and sclerotic new bone formation. In the skull, focal areas of lysis known as 'osteoporosis circumscripta' may be present.
  ii. Bowing and bending deformities are common.
- *Bone scan (Fig. 13.28B)*
  Shows increase uptake of the nucleotide in affected areas much before the radiological changes appear.
- *Histopathology*
  Shows excessive osteoclasts, irregular lamellar bone formation and replacement of the marrow by fibrous tissue.

Treatment

Treatment consists of two components:

a. *Treatment of the pathology:* This is carried out by giving drugs to decrease the increased osteoclastic activity, e.g. Risedronate along with adequate amounts of calcium and vitamin D. Exposure to sunlight is advised. Calcitonin is the drug of choice in extensive disease as well as a preoperative adjuvant.
b. *Treatment of complications*
   i. Pathological fractures are fixed. Deformities are corrected by osteotomies.
   ii. Arthritis is treated by total joint replacement when nonoperative methods fail to give relief.
   iii. Secondary osteosarcoma is treated with amputation and necessary chemotherapy and radiotherapy.

Fibrous Dysplasia

Fibrous dysplasia is a nonhereditary condition. It occurs due to a spontaneous mutation in the gene that encodes the subunit of a stimulatory G protein (Gs-α), located on chromosome 20. Due to this, there occurs a substitution of amino acid arginine by cysteine or histidine. Thus, the osteoblasts lay down fibrous tissue instead of osteoid. This results in a gradual replacement of bone by fibrous tissue (during physiologic remodeling) and development of fibrous dysplasia.

Types of fibrous dysplasia

a. Mono-ostotic; single bone is involved.
b. Polyostotic; many bones are involved.
c. McCune-Albright syndrome
   Polyostotic fibrous dysplasia, precocious sexual puberty, café au lait skin lesions.
d. Mazabraud's syndrome (Mazabraud and Girard, 1957)
   Polyostotic fibrous dysplasia with skeletal muscle myxomas.

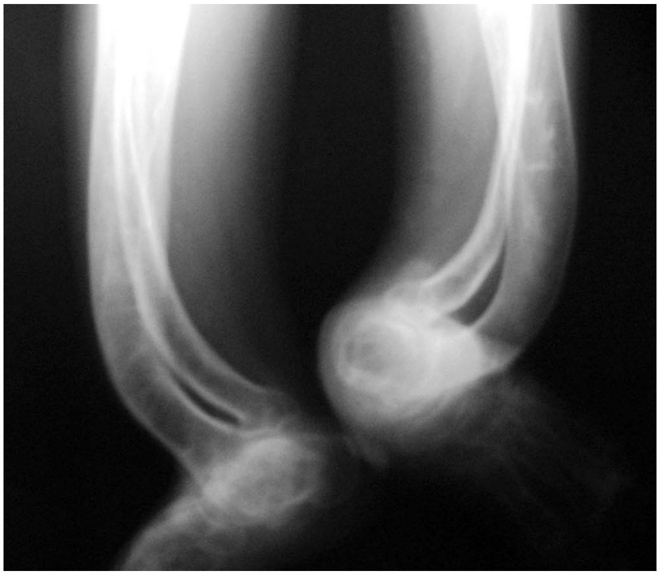


Figure 13.29

Tibia and fibula of both the legs showing the ground glass appearance and bending deformity of fibrous dysplasia.

Clinical signs and symptoms

i. Polyostotic variety presents early in childhood. Mono-ostotic variety presents as late as 30 years of age.
ii. Medical attention is sought for either bone pain, pathological fracture or the deformity.

Investigations

- X-ray—shows a typical ground glass appearance (Fig. 13.29).
- Histopathology—shows the trabeculae of woven bone embedded with fluid filled cysts. The surrounding matrix is largely fibrous.

Treatment

- *Drug therapy of fibrous dysplasia*
  Bisposphonates by their inhibitory action on osteoclasts are found to be useful. Intravenous Pamidronate is being used in pediatric age group. Supplementation of calcium and vitamin D is a must.
- *Operative treatment*
  Those who present with pathological fractures and deformities, are treated surgically with internal fixation and corrective osteotomies. Intramedullary devices should be preferred for internal fixation.

Osteogenesis Imperfecta

This condition is also known by several names some are synonyms and others are eponyms:

- Periosteal dysplasia.
- Fragilitas ossium.
- Brittle bone disease.
- Vrolik's disease.
- Lobstein's disease (1835).

The condition is hereditary and characterized by fragile bones, blue sclerae, deafness, laxity of joints and a tendency of improvement with age.

Pathology

The disease affects both endochondral and intramembranous ossification.

The initiation of osteogenesis proceeds normally up to the stage of infiltration by osteoblasts. At this stage matrix is invaded by abnormal osteoblasts which are incapable of laying down normal type I collagen to form the osteoid suitable for adequate mineralization. Very few osteocytes appear and the bone that is laid down is thin and weak.

The trabeculae formed are disorganized, sparse, thin and delicate. Thus, the stress tolerance is poor and there is a tendency for easy fracture.

Classification

Based on Shapiro's classification following types are recognized depending on the age of presentation:

I. Osteogenesis imperfecta congenita
   A. Fractures occur *in utero* or at birth. The short broad crumpled femora and ribs are characteristic.
   B. Fractures occur *in utero* or at birth. The long bone contours are normal. No chest deformities are seen.
II. Osteogenesis impefecta tarda
   A. Fractures occur after birth but before walking.
   B. Fractures occur after the child starts walking.

Based on genetic abnormality and clinical features or both, types I–IV have been described:

Type I—Dominant with blue sclera (Autosomal dominant).

Type II—Lethal perinatal with blue sclera (Autosomal recessive).

Type III—Progressive deformity. Sclerae become less blue with age (Autosomal recessive).

Type IV—Dominant with normal sclerae (Autosomal dominant).

Clinical Features

The features vary depending on the severity of the condition.

Fracture is the most common feature with which a patient presents.

1. *Fetal and prenatal form:* This is the severe form of the disease. Stillbirth and death in the early newborn period is common. Life expectancy is poor.
2. *Infantile form:* This form is less severe when compared to fetal form. If the child survives the initial few weeks, may survive for long.
3. *Adolescent form:* This is identified late in childhood and adolescence when the child sustains a fracture with a relatively trivial trauma.
   On examination following findings may be present:
   - Blue sclera.
   - Deafness.
   - Laxity of joints.
   - Short stature.
   - Broad skull.
   - Dentigerous imperfecta (poor calcification of teeth).

Investigations

- *X-ray shows:*
  - Thin bone with bulbous ends
  - Pencil line cortex
  - Thin sparse trabeculae
  - Plastic bowing may be seen
  - Fractures in various stages of healing
  - Kyphoscoliotic deformity in the spine
  - Popcorn calcification at the distal ends of long bones
- *Laboratory investigations*
  - Calcium and phosphorus are normal. Alkaline phosphatase is elevated.

Treatment

Following are the aims of treatment:

A. To strengthen the bone by giving bisphosphonates.
B. To prevent the pathological fractures.
C. To correct the deformities.

Prognosis

In lethal variety, there are no survivals. Stillbirth and perinatal death are common.

In other less severe varieties, repeated fractures are common and the child will remain stunted in growth. Deformities are commonly seen.

Revision Questions

Note: *Any of the above conditions can be asked as a short notes or a long question.*

Short notes

Write notes on:

a. Skeletal dysplasias.
b. Achondroplasia.
c. Hypochondroplasia.
d. Paget's disease.
e. Osteopetrosis.
f. Fibrous dysplasia.
g. Osteogenesis imperfecta.

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14 Miscellaneous Conditions

MALLET FINGER

Disruption of the extensor mechanism at the distal interphalangeal (DIP) joint leads to a flexion deformity. This is known as mallet finger.

Etiology

A direct blow to a finger tip which is rigidly held in extension, causes a sudden flexion at the distal interphalangeal (DIP) joint of the finger causing:

a. The rupture of the lateral slips of the extensor mechanism (Fig. 14.1).
b. An avulsion fracture of the base of the distal phalanx, e.g. a baseball or a volleyball injury (Fig. 14.2).

Clinical Features

a. The finger remains flexed at the DIP joint (distal interphalangeal).
b. There no active extension but passive extension is possible.
c. Tenderness and minimal swelling is present in acute cases.

Treatment

Soft tissue injuries are treated by mallet finger splints (Figs 14.3A to C) and immobilized for a period of three weeks followed by gradual mobilization. Different splints are available.

Avulsion fractures are fixed by using K-wires/pull-out sutures.

BOUTONNIERE DEFORMITY

Disruption of the extensor mechanism at the proximal interphalangeal (PIP) joint results in flexion deformity at the PIP joint and hyperextension at the distal interphalangeal (DIP) joint. This is known as Boutonniere deformity.

Etiology

It occurs due to the rupture of the central slip of the extensor mechanism as a result of trauma or inflammation.

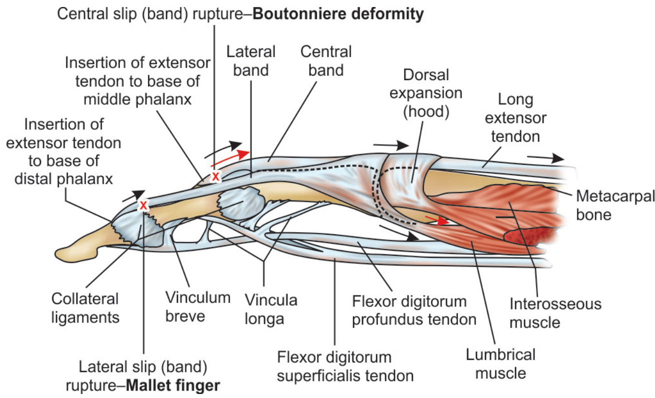


Figure 14.1

Insertion of the central slip and the lateral slips to the middle and the distal phalanx, respectively. Site of rupture for producing Mallet finger and Boutonniere deformity is marked by letter X in red.

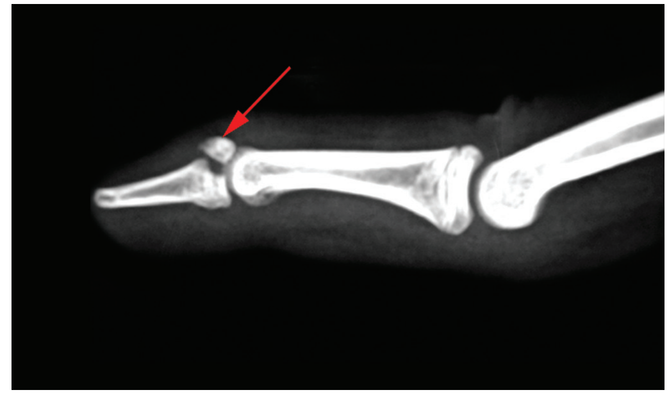
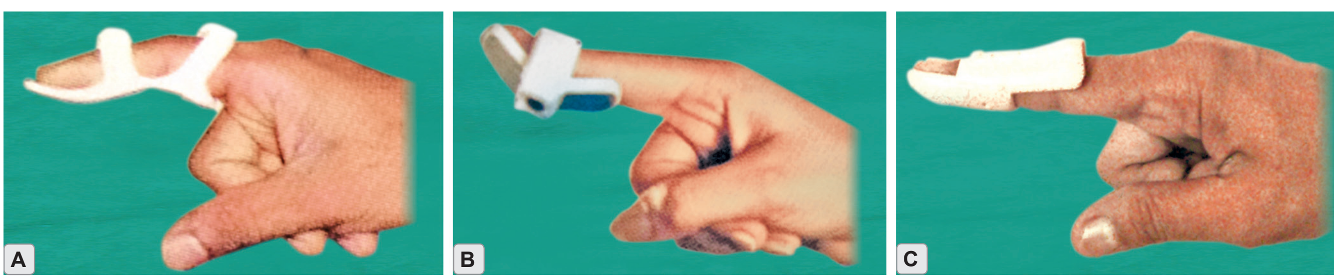


Figure 14.2

An avulsion fracture of the base of distal phalanx. The resultant deformity is a 'Mallet finger'. The patient was a goal keeper and the injury happened during a soccer match when he blocked a penalty kick. Avulsion force is exerted by the lateral slips of the extensor expansion.



Figures 14.3A to C

Different designs of mallet finger splints: (A) Frog splint; (B) DIP extension splint; (C) Stax mallet.

Trauma

In trauma, it can occur as a result of (i) direct penetrating injury, (ii) a forcible flexion at the PIP joint in a finger held firmly in extension, and (iii) in a volar dislocation of the PIP joint.

Rheumatoid Arthritis

Rupture occurs because of the synovitis occurring in the PIP joint. This slowly flexes the joint, stretches the central slip which progressively becomes thinner as a result of inflammation and stretching and finally ruptures.

Clinical Features

a. The finger remains flexed at PIP joint and extended at DIP joint.
b. No active extension at PIP joint is possible.
c. Tenderness and swelling over PIP joint in acute cases.

Treatment

Splinting is the first option in closed injuries as well as in early rheumatoid pathology.

Surgical repair and reconstruction is considered in total rupture.

TRIGGER FINGER/THUMB

It is known as trigger finger because a bent and locked finger, when straightened, opens with a snap, i.e. like a trigger. This is exactly the complaint with which a patient with trigger finger presents.

Etiology

More common in women than in men and in diabetics. Those who are involved in manual labor which involves gripping of a hard object are at a greater risk of developing trigger finger. This is the result of inflammation of a flexor tendon sheath the tendon sheath of the flexor tendon

of a finger. As the sheath gets thickened, the canal for gliding of the tendon narrows and triggering develops.

Clinical Features

a. Extension of a flexed finger is associated with pain and a snap (Triggering).
b. Tenderness over Metacarpophalangeal joint (MCP) joint of involved finger.

Treatment

It begins with nonoperative measures in mild and early cases. If symptoms are not relieved, surgical release is done.

a. Nonoperatively, treated by anti-inflammatory drugs and steroid infiltration into the flexor tendon sheath.
b. Surgically, treated by the release of the flexor tendon sheath. This relieves the symptoms permanently.

de QUERVAIN'S DISEASE

Named after Fritz de Quervain, a Swiss surgeon who described it in 1895.

Also known as de Quervain's syndrome, Washerwoman's sprain, Mother's wrist; it is an inflammation of the tendon sheath or the tunnel that surrounds the tendons of abductor pollicis longus and extensor pollicis brevis. It is commonly seen in women and is thought to be due to increased angle of the radial styloid process.

Clinical Features

Pain, swelling and tenderness on the radial side of the wrist with difficulty in gripping the object.

Finkelstein's Test

The patient is asked to make a fist. The test is performed by passively ulnar deviating the fist. This results in exaggeration of pain at the inflamed site.

Treatment

a. Nonoperatively, treated by giving rest to the involved hand using splints, administration of anti-inflammatory drugs and infiltration with steroids.
b. Surgically, treated by release of the sheath. This gives permanent relief. Superficial branch of the radial nerve is to be protected during the procedure.

DUPUYTREN'S CONTRACTURE

It is the contracture of the palmar aponeurosis which results in the flexion deformity of the fingers. Ring and the little fingers are commonly involved.

Named after Baron Dupuytren who described the condition in 1831. He attributed it to be the result of holding the reins of a horse for many years. This was not found to be true.

Structure of the Palmar Aponeurosis

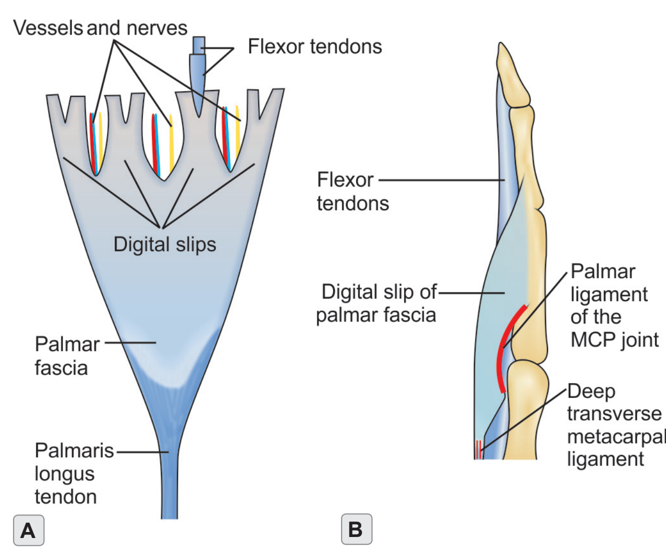
The palmar aponeurosis (fascia) is formed by a thick condensation of the deep fascia. It is triangular in shape and attached,

Proximally—to the flexor retinaculum and palmaris longus tendon.

Laterally—covers the thenar muscles of the hand.

Medially—covers the hypothenar muscles.

Distally—divides into four major slips. Each of the major slip bifurcates into two and gets attached to the transverse metacarpal ligament, the fibrous flexor sheath and along the sides of the entire proximal phalanx and proximal part of the middle phalanx of each digit. It is not attached to the distal phalanx (Figs 14.4A and B). Hence, in Dupuytren's contracture, the



Figures 14.4A and B

Palmar aponeurosis, its division and attachment to the deep transverse metacarpal ligament. The digital vessels and nerves, the lumbricals and the flexor tendons emerge from underneath the slips and enter the digits.

distal interphalangeal joint does not develop flexion contracture but remains extended.

Etiology

It shows a genetic predisposition in some. Diabetes, epilepsy and alcohol dependency are the other predisposing factors.

Clinical Features

a. Thickened fibrous bands felt over the ulnar side of the palm. Sometimes, nodular thickening is felt.
b. Tenderness over the involved area at the base of the digits.
c. Flexion deformity at the MCP and PIP joints of involved fingers.

Treatment

Minor deformities are treated nonoperatively and patients are advised to avoid the predisposing factors, e.g. alcohol intake.

In those cases where the contracture is severe and painful, surgical excision of the contracted tissue is advised.

CARPAL TUNNEL SYNDROME

It is the compression of the median nerve in the carpal tunnel causing numbness in the hand. If left untreated paralysis of the thenar muscles takes place over a period of time. Rarely, severe sensory disturbance and trophic ulcers can develop (Fig. 14.5).

Attachment of flexor retinaculum:

Medially

i. Pisiform
ii. Hook of hamate

Laterally

i. Tubercle of scaphoid
ii. Crest of trapezium

Etiology

Compression occurs due to increase in volume of the contents of the canal or decrease in available space in the canal. Well identified causes are as follows.

a. Space-occupying lesions in the canal such as ganglion, tenosynovitis, lipoma, etc. increase the volume of the contents in the canal and cause compression.
b. Malunited Colles' fracture, degenerative and inflammatory arthritis of the wrist makes the canal non-congruous and decrease the available space in the canal thereby causing compression.
c. Physiological cause is pregnancy. The compression is transitory which gets relieved after delivery.

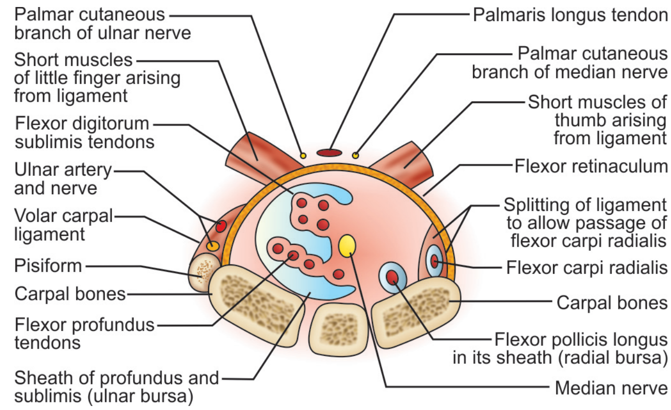


Figure 14.5

Structures under the flexor retinaculum and the boundaries and the contents of the carpal tunnel.

Clinical Features

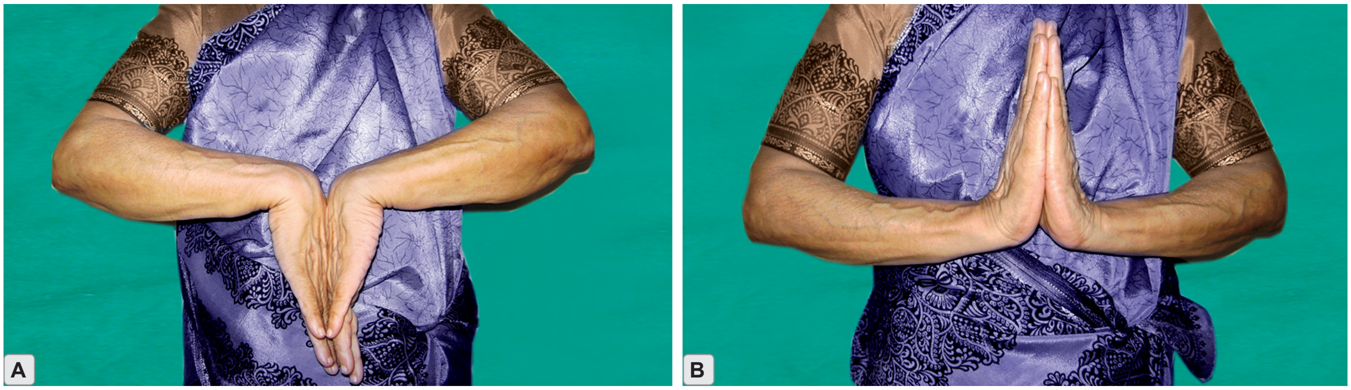
a. Patient presents with diminished sensation and pain along the distribution of the median nerve in the hand. Pain begins initially at night. Later on, becomes more constant and interferes with daily activities.
b. Phalen's test positive (Figs 14.6A and B).
c. Tinel's sign positive.
d. Nerve conduction velocity shows delay in conduction.
e. Ape thumb deformity if paralysis of abductor pollicis brevis (APB) occurs.

Treatment

Nonoperative treatment is indicated

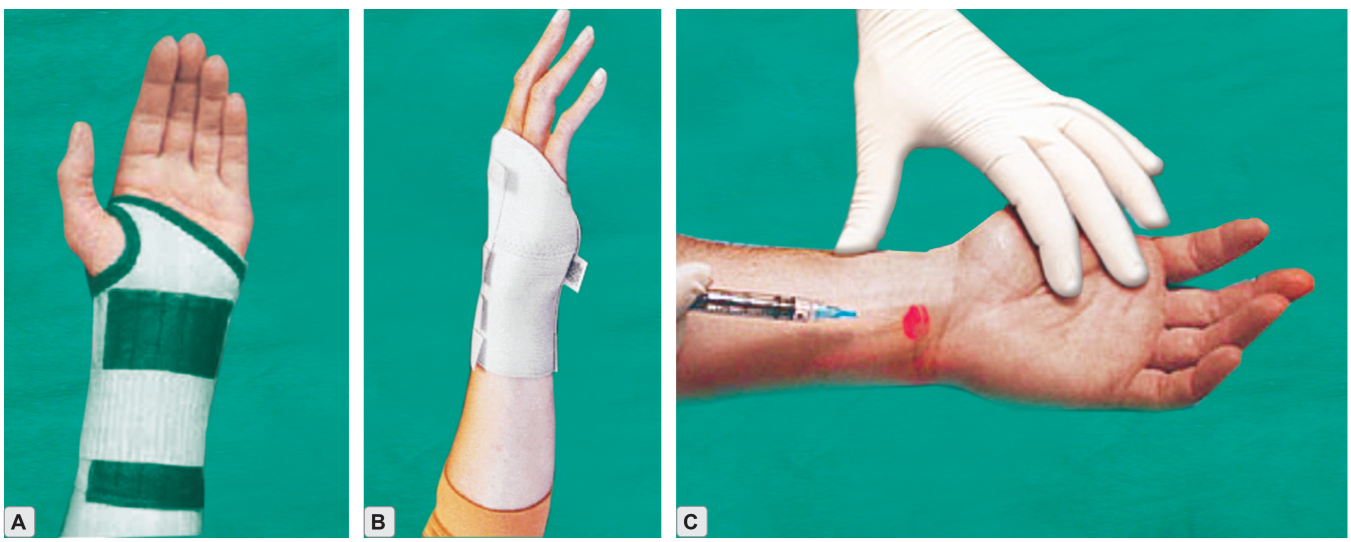
i. If the compression is physiological and no pathological cause is observed (Figs 14.7A to C).
ii. In early cases with transitory compression.

Measures undertaken are rest to the part, supportive splints and anti-inflammatory drug therapy. Infiltration of the tunnel with steroids can also be tried. If no relief is obtained, surgical decompression is undertaken by dividing the flexor retinaculum at the wrist.



Figures 14.6A and B

(A) Phalen's test; (B) Reverse Phalen's test. In both the tests, with elbow and wrist in 90°, a firm pressure is excreted to opposing hands.



Figures 14.7A to C

(A and B) Supportive splints for carpal tunnel syndrome; (C) Steroid injection into the carpal tunnel.

When space occupying lesions are detected, the tunnel has to be explored after dividing the flexor retinaculum. The lesion has to be excised and the tunnel is deroofed by not suturing the divided flexor retinaculum.

Decompression is also indicated when mechanical alteration of the tunnel has occurred.

TENNIS ELBOW

It is also known as lateral epicondylitis. It occurs due to repetitive stretch of the common extensor origin at the lateral epicondyle. Common in tennis players and symptoms are observed especially during a back hand stroke. Hence, the name was derived. It is also common in manual laborers, e.g. plumbers, gardeners, etc.

Pathology

Repetitive stretch causes microtears resulting in inflammation, degeneration and fibrosis of the common extensor origin there by making it less elastic. Extensor carpi radialis brevis is commonly affected (Fig. 14.8).

Clinical Features

a. Pain and discomfort in the region of common extensor origin, especially while lifting heavy objects.
b. Tenderness over the same area (Fig. 14.8).

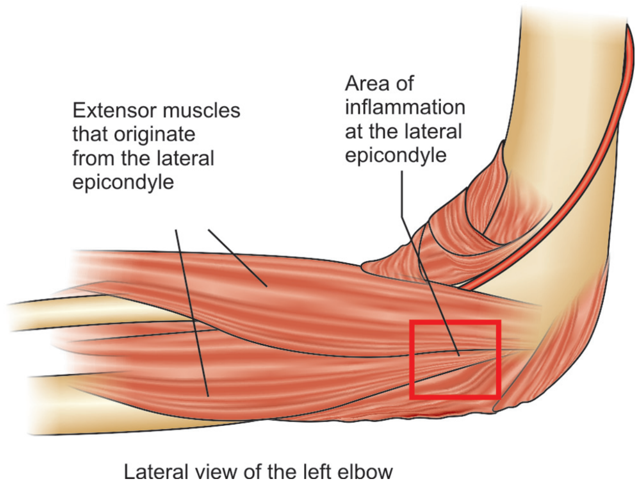


Figure 14.8
Common extensor origin of muscles at the lateral condyle.

Figure 14.10
Tennis elbow strap.

Figure 14.9
Cozen's test. The forearm should be pronated fully and then only the wrist is actively dorsiflexed against resistance.

Test

Tennis elbow is diagnosed by a specific test known as Cozen's test (Fig. 14.9). The patient is asked to hold the upper limb with elbow in flexion and the forearm in full pronation. Then asked to dorsiflex and radially deviate the wrist while resistance is applied. Severe pain is observed at the common extensor origin.

Treatment

a. Avoid repetitive stretch by not performing such activities which cause pain and discomfort till healing occurs. This is followed by gradual resumption of activities.
b. Wearing supportive splints and straps for tennis elbow (Fig. 14.10).
c. Ice packs for acute episode and heat therapy for chronic.
d. Anti-inflammatory drugs.
e. Corticosteroid injections.
f. Exercise therapy and occupation therapy.
g. Surgery in refractory cases (Rarely done).

GOLFER'S ELBOW

It is known as medial epicondylitis and commonly seen in golf players. Hence the name. It results due to chronic repetitive stretch of the common flexor origin at the medial epicondyle. Pathology is similar to that of tennis elbow.

Test

By asking the patient to hold the limb straight with the elbow in extension. Then, he is asked to make a fist and flex the wrist while resistance is applied. Patient complains of severe pain in the region of medial epicondyle.

Treatment

Same manner as in tennis elbow.

SUPRASPINATUS TENDINITIS

It is an inflammation of the supraspinatus tendon due to minor degrees of trauma. It results in difficulty to initiate abduction. Diagnosed by the presence of a tender spot over the tendon and difficulty in initiation of abduction.

Treatment

Treatment is nonoperative. Measures taken are rest to the part, administration of anti-inflammatory drugs and local cortisone injections.

Current concept is that the condition is a part of the shoulder impingement syndrome and it is the impingement of the supraspinatus tendon which leads to inflammation (Refer Painful Arc Syndrome).

Calcific Tendinitis

It was Codman who first identified calcification in the tendons of the rotator cuff. The exact cause is not known. It is thought that poor blood supply and attrition creates a conducive environment for calcification (Figs 14.11 and 14.12).

Clinical Features

a. Patient presents with acute onset of pain, worse at night along with limitation of abduction of the shoulder. An X-ray confirms the diagnosis.
b. Severe tenderness over the greater tuberosity.
c. Active shoulder movements are limited.

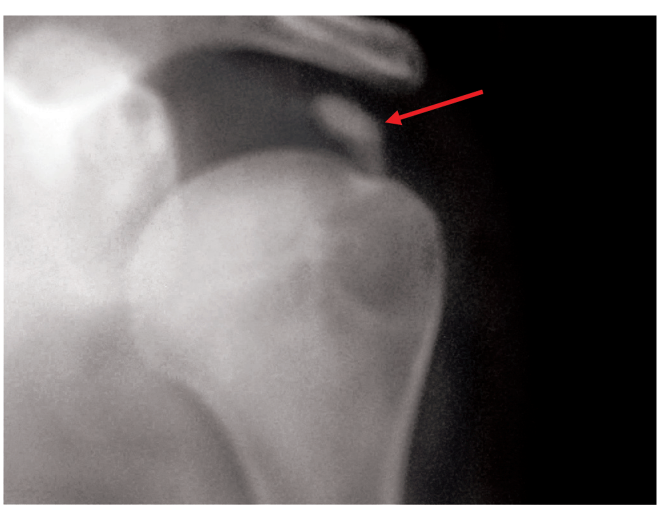


Figure 14.11

Uniformly dense deposit. This indicates the acute phase of calcific supraspinatus tendinitis.

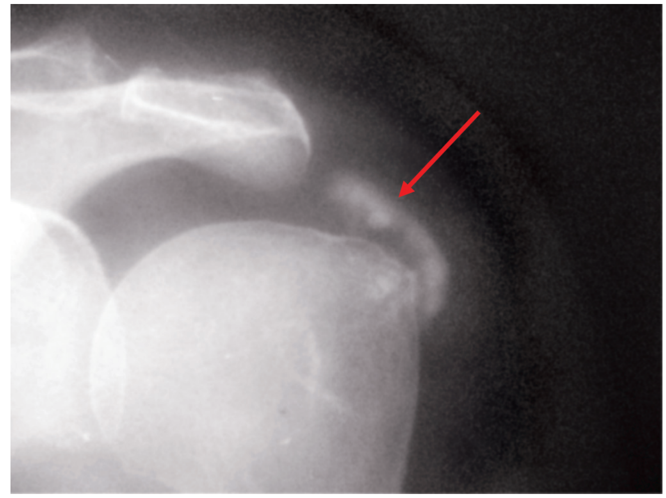


Figure 14.12

Amorphous calcific deposit. The amorphous nature indicates resolution phase of the lesion.

Treatment

a. Ice packs to the shoulder.
b. Anti-inflammatory drugs.
c. Corticosteroid injections.
d. Large deposits which do not respond to nonoperative methods are excised by open/arthroscopic surgery.

SUBACROMIAL BURSITIS

Subacromial bursa is situated in the shoulder between the coracoacromial ligament, acromion, coracoid and deep portion of the deltoid muscle superiorly and supraspinatus tendon inferiorly. It helps in smooth gliding of the tendon of supraspinatus during movements of the shoulder and protects it form damage.

Inflammation of this bursa results in subacromial bursitis.

Etiology

a. Primary
   Occurs rarely. Seen in autoimmune diseases, e.g. rheumatoid arthritis, crystal synovitis, infection, etc.
b. Secondary
   Occurs as a result of local pathology around the shoulder, e.g. rotator cuff tear and shoulder instability.

Clinical Features

The features closely mimic rotator cuff injury and are difficult to differentiate. In both, abduction against resistance, results in pinching and pain as the deltoid contracts. However, if a caudal glide or traction is applied during abduction, the space between the coracoacromial arch and the tuberosity of the humerus increases and pain of bursitis may decrease. But, this may not happen in rotator cuff injuries.

Investigations such as ultrasound and MRI detect accumulation of fluid in the bursa and are confirmatory in the diagnosis of subacromial bursitis.

Treatment

Nonoperative

Subacromial bursitis responds very well to nonoperative treatment.

Rest, anti-inflammatory drugs, steroid infiltration, cold therapy in acute cases, ultrasound therapy and heat therapy in long-standing cases, gives relief of symptoms and cure.

Operative

Surgery is reserved for resistant cases which do not respond to nonoperative measures. Excision of the bursa is done by open or arthroscopic method. Arthroscopic excision has a distinct advantage of visualizing other structures, there by enabling the surgeon to manage other pathologies when found, e.g. rotator cuff tear, spurs, etc.

PAINFUL ARC SYNDOME

Other Names

Impingement syndrome, supraspinatus syndrome, 'Swimmer's syndrome' and 'Thrower's shoulder'.

It is a clinical syndrome characterized by pain in the shoulder during an arc of movement between 60° and 120° of abduction.

Etiology

a. Increase in bulk of the contents in the subacromial space, as seen in inflammation of the rotator cuff, subacromial bursa and calcification of supraspinatus.
b. Decrease in the size of the subacromial space, as seen in acromioclavicular arthritis, variations in the acromion, thickening of the coracoclavicular ligament, etc.

Clinical Features

a. Pain, weakness and a loss of movement at the affected shoulder.
b. Pain worsened by overhead movement.
c. Pain worsened by lying on the affected shoulder.
d. Grinding or popping sensation during movement may be experienced in some cases.

Treatment

Treatment depends on the pathology that is causing impingement.

Nonoperative

Measures are same as that described for subacromial bursitis.

Operative

Procedures aim at enlarging the subacromial space and decompressing it.

Shaving of the spur (when present), excision of the impinging acromion, etc. are examples of the surgical procedures that are done for 'Impingement syndrome'.

PERIARTHRITIS SHOULDER

It is also known as adhesive capsulitis and frozen shoulder. Also referred to as the 'shoulder of fifties' because the condition is very common around that age. It is common in diabetics.

It presents with gradual painful restriction of all the movements of the shoulder. It is thought to be due to an inflammatory degeneration of the shoulder joint capsule and the soft tissues surrounding it, resulting in adhesions.

Clinical Features

a. Pain of insidious onset. Worse at night. Increases on movement.
b. Gradual onset of painful limitation of all the movements, abduction and external rotation in particular.
c. Tenderness all round the shoulder with more than one tender spot.

X-ray—shows rarefaction of the head of the humerus. Degenerative changes may be seen in acromioclavicular joint.

Treatment

a. Anti-inflammatory drugs.
b. Gradual and active mobilization of the shoulder.
c. Physiotherapeutic application of moist heat therapy, ultrasound therapy, etc.
d. Manipulation under anesthesia followed by exercise therapy.
e. Infiltration with corticosteroids.

Natural course of the disease: Slow recovery occurs in 2 years.

Current Concept

There is nothing like an entity known as Periarthritis. The shoulder stiffness is the result of some derangement that has occurred either in the bone and joint component or in the soft tissue component of the shoulder. Unless this is diagnosed and treated the stiffness does not improve and shoulder does not become normal. At times, it may not be possible to determine the exact cause.

In other words, immobility is the result of an injury/insult. The injury might have healed leaving behind stiffness. Hence, a proper history, careful examination followed by investigations and diagnosis is essential while managing the so called periarthritis which is a general term and not an entity by itself.

Note: *The topic 'Periarthritis' has been retained only for the sake of clarification.*

CALCANEAL SPUR

It is an ossification/calcification occurring at the insertion of the plantar fascia to the periosteum, on the undersurface of the calcaneus (Fig. 14.13).

Etiology

Repetitive stress (especially unprepared for) results in stretching of the plantar fascia and microtears. Ultimately, calcium gets deposited and a spur develops. Thus, spur is the result of the inflammation. The symptoms are due to the inflammation and are present during the early process of spur formation and not after the spur formation is complete. This is proved beyond doubt because many spurs are found as an incidental finding which are totally asymptomatic. Whereas, heel pain can occur without the presence of a spur.

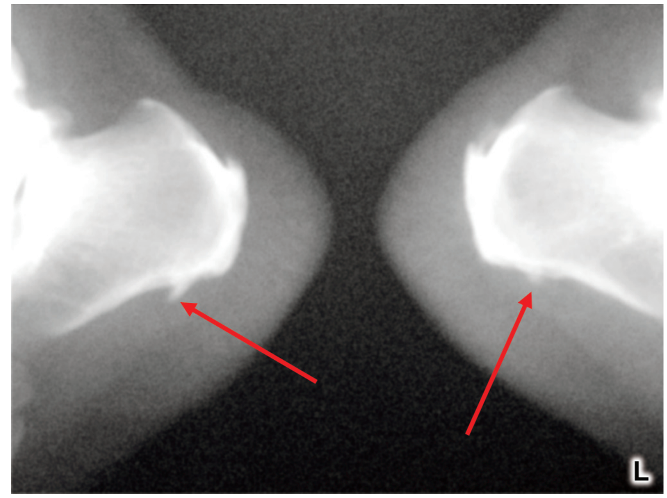


Figure 14.13

Bilateral calcaneal spur. See the sharp pointed nature of the spur on the right side and amorphous appearance on the left side. The spur on the right side was asymptomatic. It was the spur on the left side, which is just developing, that was painful.

Clinical Features

a. Pain on the undersurface of the heel which is relieved by rest and starts again at the initiation of activity.
b. Classically worsens when a person gets up in the morning.
c. Aggravated by walking on a hard surface.

Treatment

Always the treatment is non-operative. Over a period of time the symptoms subside. The condition is managed by:

a. Anti-inflammatory drugs.
b. Soft footwear/cushioned heel.
c. Physiotherapeutic measures.
d. Reducing unaccustomed stress.
e. Corticosteroid infiltration.

GANGLION

Other names: 'Bible bumps' or 'Gideon's disease'

Note: *In the past, heavy Bible was used to hit and rupture the cyst as a method of treatment. Hence, the name 'Bible bumps' was derived. Currently, rupturing the cyst is rarely practiced.*

It is a benign, cystic swelling containing thick, gelatinous fluid, occurring near the joints and in the tendon sheaths.

Age: Any age group. Common in young. Not very common in elderly.

Sex: More common in women than in men.

Site: In the hands or in the feet; originating in the vicinity of joints or in the tendon sheaths.

Pathology: Leaking of synovial fluid from the joint forms a cystic swelling extracapsularly. The fluid does not flow back into the joint because of a check valve like mechanism operating. Thus, the fluid that accumulates becomes glairy (slimy viscid) and remains as a cystic swelling. Accumulation of more fluid increases the size of the cyst.

This postulate appears to be true because the cysts near the joint invariable show connection to the joint.

Histopathologically, the cyst has a fibrous wall without any lining of specialized cells and contains glairy material.

Treatment

Nonoperative

a. Firm pressure of an elastic band/strap at accessible sites is known to cause regression of the cyst in about 4–6 weeks.
b. Aspiration of the cyst and injection of hyaluronidase along with long acting steroids (Methyl prednisolone depot preparation) is known to cause regression in some cases, especially in those which arise from the tendon sheath.

Operative

Surgical excision is indicated mainly for cosmetic reasons, pain and interference in the movement of the joint. Both open and arthroscopic excision is practiced.

Rate of recurrence after surgery is about 5–10%. The check valve like mechanism has to be excised during surgical excision to prevent recurrence.

BAKER'S CYST

This is named after a British surgeon William Morrant Baker (1839-1896). He described this in the year 1877 and gave the name 'Baker's cyst' (Fig. 14.14).

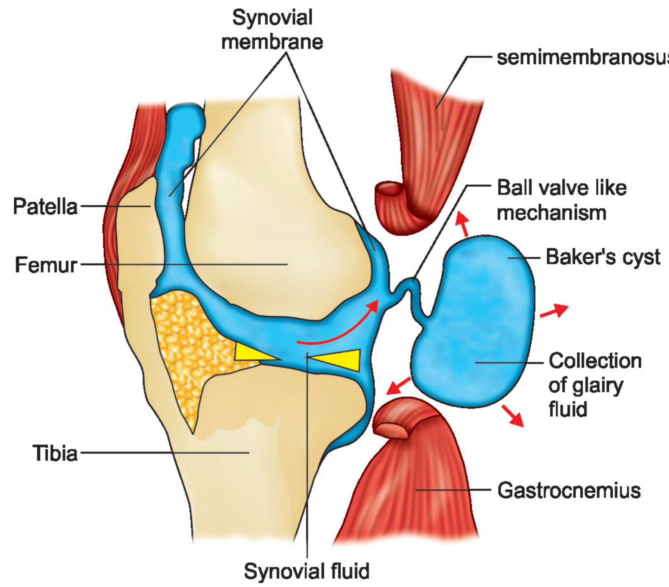


Fig. 14.14
Bakers cyst.

It is a benign invagination of the synovial membrane of the knee, posteriorly, in the popliteal region, presenting between the medial head of the gastrocnemius and the semimembranosus, as a cyst. The cyst contains synovial fluid. Any form derangement, e.g. arthritis, cartilage tear, etc. can cause this invagination, resulting in a cyst formation. The cyst contains synovial fluid and communicates with the joint.

Treatment

a. Asymptomatic cysts do not require treatment.
b. Ruptured cysts are painful and are treated by rest, cold packs to the region, limb elevation, anti-inflammatory drugs and steroid infiltration into the joint to reduce inflammation.
c. Symptomatic cysts can be treated with aspiration and hydrocortisone injection. This reduces the size of the cyst and the symptoms.
d. Surgical excision of the cyst is indicated when there are pressure symptoms on the surrounding structures, pain and discomfort.

Important Differential Diagnosis

1. Aneurysm of the popliteal artery.
   Following are the differentiating points.
   a. Baker's cyst is not pulsatile whereas the aneurysm is pulsatile.

b. A Baker's cyst is transilluminant whereas the aneurysm is not.

2. Semimembranosus bursitis
Semimembranosus bursitis is seen medially. Whereas Baker's cyst is seen in the midline.

NEUROPATHIC JOINT

Also known as Charcot's joint, named after Charcot, who first described this condition in a case of 'tabes dorsalis' in the year 1868. Steindler was the first person to observe a destructive atrophic form and a hypertrophic proliferative form of this disease (Fig. 14.15).

Etiology

The cause is neuropathia occurring secondary to:

i. Tabes dorsalis
ii. Hansen's disease
iii. Diabetic neuropathy
iv. Syringomyelia
v. Peripheral nerve lesions
vi. Hereditary sensory neuropathy

Figure 14.15

Classical changes of Charcot's joint. Note extensive degeneration and disorganization of ankle, subtalar, tanonavicular and calcaneocuboid joints. Shape of the talus is totally altered.
New bone formation seen, is an attempt at healing of fractures. In spite of such severe alterations, the joint was totally painless.

Pathology

The pathological process begins with an injury. A single injury heals uneventfully even in a neuropathic joint when diagnosed and treated adequately. Animal experiments have proved this fact beyond doubt. It is the repetitive trauma which is known to initiate the degenerative process. Though the joint lacks sensation, it responds to injury by causing inflammation. The joint shows all the signs of inflammation such as calor, rubor and tumor, except dolor, which is absent. This is the initial destructive atrophic phase of the pathology where there is hyperemia. Hyperemia is necessary to clear the debris. Since patient does not experience pain, there is a likelihood that the initial injury is neglected and this injured hyperemic joint is exposed to repetitive stress causing further injury and a vicious cycle sets in. Thus, the initial inflammatory reaction that had set in becomes persistent and prolonged. This causes progressive degeneration and formation of debris. The joint in this stage is badly swollen, shows increased warmth and redness. If the joint is not protected at this stage, it leads to total destruction and disorganization. If the joint is protected, the process of repair begins. Repair results in the formation of dense fibrous tissue in the surrounding soft tissue and dense sclerotic bone on either side of the joint. Massive callus may form in the soft tissue. Lipping may develop at the margins of the joint. This is the hypertrophic, proliferative form of the disease which is the phase of repair. The signs of inflammation decrease and no calor, rubor and tumor are observed during this phase.

Clinical Features

There is definite history of having neuropathic problem.

a. A badly swollen joint due to hemarthrosis/ effusion.
b. Signs of inflammation present in the acute phase.
c. Lack of pain is the classical feature.
d. Exaggerated movements and laxity is seen in later stages.
e. Deformity and total disorganization is seen in the final stage.

Diagnosis

X-ray—shows classical features of a neuropathic joint with destruction and disorganization out of proportion to the clinical symptoms (Fig. 14.15).

Treatment

The proverb "*Prevention is better than cure*" is very apt for neuropathic joint. Inadequately protected injuries of the joint, are the ones which are responsible for destruction.

a. Protection of the joint, is of great importance when neuropathia is suspected, in order to prevent the onset of the degenerative process.
b. Once the changes have taken place, the aim of treatment is to prevent further degeneration and damage. This is achieved through occupational guidance and advocating protective splints and calipers. Joint aspiration may be indicated to empty the collected fluid. Protection should be continued till warmth and redness subsides. This is followed by measures which prevent further injury, during activities of daily living.
c. Arthrodesis is done for a totally destroyed and disorganized joint. Arthrodesis should never be done during the acute inflammatory phase. It should always be attempted after healing has occurred. X-rays help to confirm healing. The dense sclerotic bone has to be excised till the raw bleeding bone is reached, in order to achieve sound fusion.
d. When the joint is badly infected amputation may have to be considered.

CONGENITAL TORTICOLLIS OR WRY NECK

It is a condition seen at birth, due to fibromatosis of the sternomastoid muscle. The incidence of associated DDH is reported to be from 7 to 20%.

Natural Course of the Fibromatosis

It manifests as a palpable tumor at birth, which attains a maximum size within 1–2 months and then, starts regressing. If it does not disappear within an year, fibrosis occurs which results in a fixed deformity.

Clinical Features

a. Inclination of the head to the same side and turning of the face to the opposite side.
b. Elevation of the ipsilateral shoulder.
c. Decrease in fronto-occipital diameter of the skull.

Diagnosis

It is not difficult to diagnose torticollis. Diagnosis is simple and is made by inspection, confirmed by palpation (sternomastoid tumor) and elicitating limitation of movements of the neck.

Treatment

Treatment should start at birth. Up to 1 year of age, the child is managed nonoperatively by advocating passive stretching and maintaining the position in sleep.

Role of Surgery

Surgery is done only when the nonoperative treatment fails. Ideal age of surgery is at 1 year. Surgery should never be done in infancy as there is good chance of regression with growth and nonoperative treatment.

Type of Surgeries

i. Release of the clavicular end of the muscle.
ii. Release at both the clavicular and the mastoid end.
iii. 'Z' lengthening.

Prognosis

Prognosis is good. The deformity is fully correctable when the surgery is done early (at 1 year) before the deformity gets established.

IDIOPATHIC AVASCULAR NECROSIS OF THE HEAD OF THE FEMUR

Other Names

- Aseptic necrosis
- Osteonecrosis
- Ischemic bone necrosis

It is a condition of unknown etiology occurring between the age of 20 and 40 years. Men are more commonly affected than women.

Clinical Features

a. Begins with dull aching pain in the hip.
b. Pain progressively becomes severe and painful limitation of movement develops.
c. Fixed deformities develop in later stages.

Staging: Ficat and Arlet (1980)

(Based on this classification 4 stages are identified)

Stage I: Shows no X-ray changes and diagnosis is made by measurement of intraosseous pressure and bone biopsy.

Stage II: The femoral head contour is normal but there are early signs of reactive changes in the subchondral area. Crescent sign (crescent-shaped area of increased density in the subchondral region) is seen.

Stage III: Clear cut X-ray signs of osteonecrosis with evidence of structural damage and distortion of the bone outline.

Stage IV: There is collapse of the articular surface and signs of secondary osteoarthritis (OA).

These days MRI is used in the early diagnosis (a band like low intensity signal on the T1-weighted image is the first indication). Also the disease is staged based on MRI findings.

Radiological Features of AVN

- Increased density of the subchondral bone.
- Maintenance of joint space until late period of the disease. This is the classical feature which is in contrast to osteoarthritis and rheumatoid arthritis.
- Secondary osteoarthritic changes in advanced stage.

Treatment

Stages I and II: Femoral core decompression with or without fibular strut grafting.

Stages III and IV: Total hip replacement.

Known causes of AVN of femoral head:

Traumatic causes
- *Fracture neck of femur/ femoral head*
- *Dislocation around hip*
- *Slipped capital femoral epiphysis*

Nontraumatic causes
- *Massive arterial occlusion*
- *Perthes' disease*
- *Venous thrombosis*
- *Alcoholism*
- *Hypercortisonism/steroid intake*
- *Caisson's disease*
- *Sickle cell disease*
- *Gaucher's disease*
- *Infections*
- *Familial hyperlipidemias*

SPONDYLOLISTHESIS

Spondy—means Spine. Olisthesis—means slipping in Greek language. Thus, the word Spondylolisthesis refers to slipping of one vertebra over the other. This condition was first described by a Belgian obstetrician Dr Herbinaux in the year 1782, when he found a bone anterior to the sacrum, obstructing the vagina.

For a slip to occur a defect should develop in the stabilizing structures namely the articular facets, the neural arch or the normal bony structure. The lumbosacral area is the common site where this slip occurs. The slip in majority of the cases is in a forward direction, because of the lumbar lordosis. When the slip is backwards, it is known as retrolisthesis.

Types: Based on Wiltse Classification (Figs 14.16A and B)

i. Dysplastic (congenital)
ii. Isthmic (defect/spodylolysis in pars interarticularis)
iii. Degenerative
iv. Traumatic
v. Pathologic

Etiopathogenesis

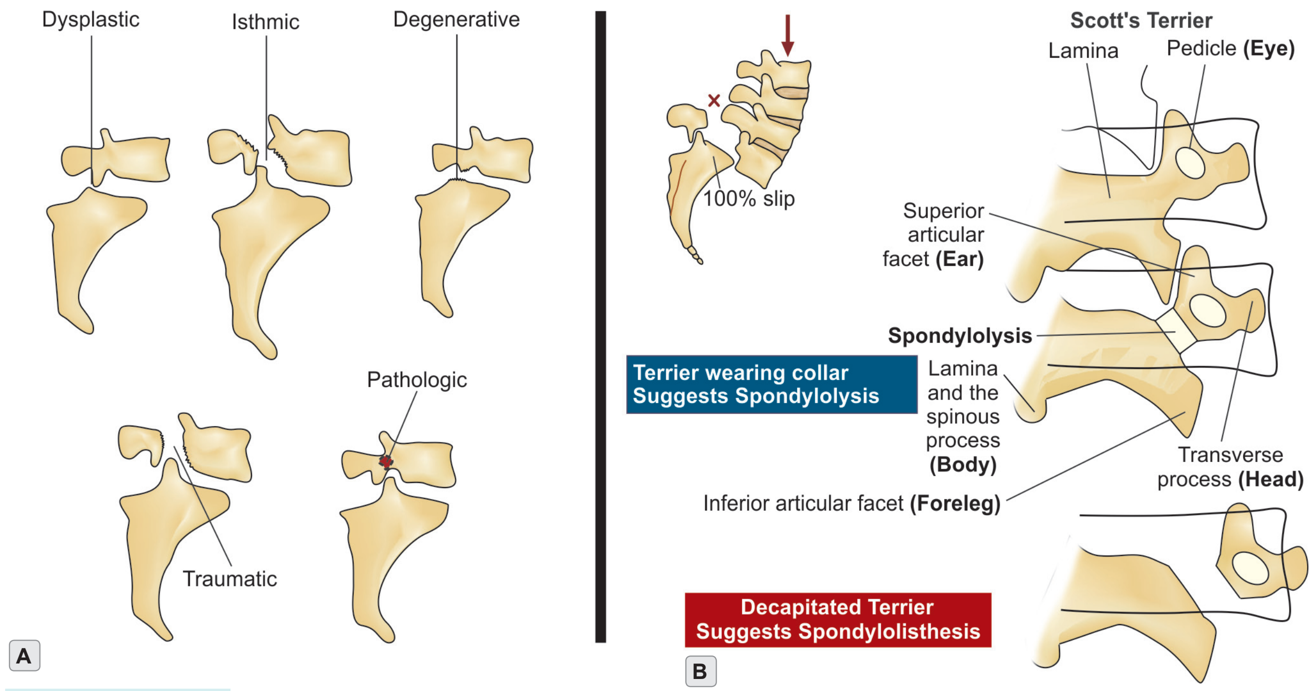
Defect in any of the stabilizing structures can lead to listhesis. Sometimes, there can be more than one pathology.

In dysplastic variety, there is agenesis or hypoplasia of the facets. This leads to the slip. It is one of the severe forms of listhesis.

In the isthmic variety, there is a defect in pars inter-articularis [Pars—means Part, Inter—means between and articularis—means articular components (Facet)]. It refers to the part between the superior and the inferior articular facets. When there is defect (which is also known as spondylolysis) in this, area two segments develop in the neural arch.

a. A posterior segment consisting of the spinous process, lamina and the inferior articular facet.
b. An anterior segment consisting of the pedicle transverse process and the superior articular facet.

With stress, the anterior segment along with the vertebral body and the column slips forwards whereas the posterior segment remains connected to the sacrum.



Figures 14.16A and B

Different types of spondylolisthesis and radiographic picture of Scott's Terrier.

In degenerative variety the degeneration involves the facetal articulation and results in instability and slip.

Traumatic type of slip results when there is trauma to the facets or the fracture of pars interarticularis.

Pathologic type is seen when the facets and the pars interarticularis are involved and destroyed in a neoplasia or infection like tuberculosis.

Traumatic and pathologic types are very rare.

Grading of the severity of the slip:

Based on Meyerding grading system four grades are recognized.

Grade 1 0–25% slip.

Grade 2 25–50% slip.

Grade 3 50–75% slip.

Grade 4 75–100% slip.

Beyond 100% the term 'spondyloptosis' is used to denote total dissociation from the lower vertebra.

Clinical Features

Backache and varying grades of neurologic deficits are the main symptoms and signs. They are directly proportional to the severity of the slip. Flattening of the buttocks and prominence of the loin crease are the local findings. The deformity becomes more pronounced as the pathology progresses and a step may be palpated at the site of listhesis.

Diagnosis

X-ray—Confirms the diagnosis.

CT and MRI—Delineate the pathology better especially when there are neurological signs.

Treatment

Aim of treatment is to prevent further slip.

Nonoperative

It is indicated only in minor grades of slip with little or no neurological signs.

Measures include modification of activity, refraining from strenuous activity, back exercises, spinal corset for support, drug therapy and regular follow-up to monitor progression.

Operative

This treatment is always indicated in severe forms with neurological deficit. Decompression and spinal fusion are achieved with or without reduction of the slip and further slip is prevented. In degenerative type, decompression may be necessary.

BURSA AND BURSITIS

Bursa is a sac of fibrous tissue lined by synovial membrane, containing synovial fluid. Generally, they are situated around joints between the bone and the insertion of a tendon. Sometimes, a bursa develops at a place which is subjected to constant stress. This is known as adventitious bursa, e.g dorsum of the foot in neglected CTEV. When the bursa gets inflamed, the condition is known as bursitis.

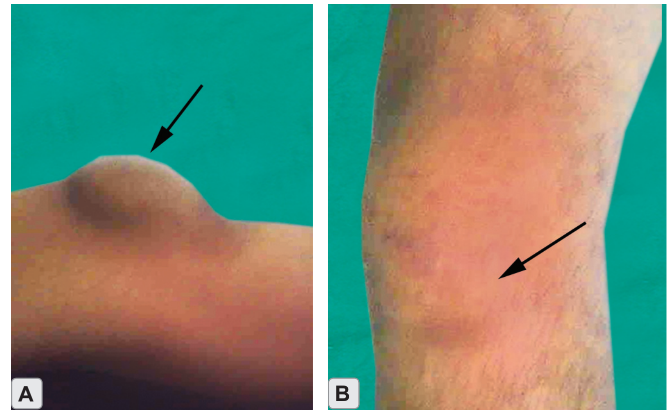
Common sites of bursae in the human body:

A. Around the shoulder
   i. Subdeltoid
   ii. Subacromial
   iii. Subscapular
   iv. Under the infraspinatus
   v. In the synovial sheath of the long head of biceps

B. Around the elbow
   i. Two in relation to triceps, at its insertion. One at the upper part of the olecranon and the other at the lower part.
   ii. Two in relation to the biceps at its insertion to the radial tuberosity.

C. Around the hip
   i. *Posterior:* Four under the gluteus maximus, one under the gluteus medius and one under the gluteus minimus.
   ii. *Anterior:* One under the psoas tendon (This might communicate with the hip joint).

D. Around the knee
   i. *Anterior:* There are four bursae anteriorly, namely, a suprapatellar, a prepatellar and two infrapatellar. One between the patellar tendon and the tibia and the other between the tibial tuberosity and the skin.
   ii. *Posterior:* There are two bursae posteriorly, one each between the heads of the gastrocnemius and the capsule of the knee joint.
   iii. *Medially:* Two bursae are present medially, one between the Sartorius, gracilis, semitendinosus and the tibial collateral ligament and the other between the semimembranosus and the tibial colateral ligament.



Figures 14.17A and B

(A) Prepatellar bursitis (Housemaid's knee); and (B) Infected infrapatellar bursitis (Clergyman's knee).

   iv. *Lateral:* There are three bursae laterally. One between the fibular collateral ligament and the biceps, the second between the fibular collateral ligament and the popliteus and the third is between the popliteus and the lateral femoral condyle.

Some of the names of commonly seen bursitis:

- *Housemaid's knee:* Prepatellar bursitis (Fig. 14.17A).
- *Clergyman's knee:* Infrapatellar bursitis (Fig. 14.17B).
- *Tailor's ankle:* Adventitious bursitis in the region of lateral malleolus.
- *Porter's shoulder:* Adventitious bursitis in the region of lateral end of clavicle and the skin.
- *Weaver's bottom:* Adventitious bursitis between gluteus maximus and ischial tuberosity.

Treatment of Bursitis

Treated with rest and supportive bandage along with anti-inflammatory drugs, if the cause is aseptic inflammation.

If the cause is sepsis, along with anti-inflammatory drugs, adequate antibiotic therapy is necessary. This is followed by excision of the sac.

MYOSITIS OSSIFICANS

It is a condition where in ossification/calcification develops in the muscles following injury/trauma. Hence, derived the term 'Myositis Ossificans traumatica'.

Predisposing Factor

Premature return to activity following injury especially when associated with passive stretching and massage.

Pathophysiology

The basic mechanism for ossification is altered differentiation of fibroblasts into osteoblasts. This occurs following soft tissue injury and odema. The initial foci of calcification which develop in the soft tissue hematoma, proceed to maturation and end in ossification. Stiffness of the muscles with limitation of joint movement is the end result.

Common Sites

a. Brachialis muscle–in injuries around the Elbow.
b. Gluteal muscles—in injuries around the Hip.
c. Adductor muscles of the thigh. (also known as Prussian's Disease)
d. Quadriceps.

Treatment

Majority of the time, the treatment is non operative in the form of rest, splinting and anti-inflammatory drugs. Once the acute phase subsides, gradual and active mobilisation is advocated.

Surgical excision of the mass is done only after the maturation of the mass, if the mass interferes with the movement of a joint or a peripheral nerve is involved. Result of surgical excision is uncertain.

Surgery should never be done during the active phase of Myositis.

Note: *Any of the above can be asked as short notes.*

15 Fractures in Children

- *Physeal injuries*
- *Structure of a physis*
- *Diagnosis and management*
- *Other injuries*
- *Common fractures in the lower limb: Diagnosis and management*

INTRODUCTION

Pediatric skeleton is a growing skeleton. It differs from an adult mature skeleton in many ways. Hence, the response to injury is different from an adult bone with respect to nature of deformation and healing. Healing occurs faster, which emphasizes the need for an early management of these injuries. Delay in management, increases the difficulty in reduction and promotes development of deformities and abnormalities in growth. So,it is absolutely necessary to understand these facts before a pediatric fracture is managed (Table 15.1).

TYPES OF INJURIES

i. Physeal injuries
ii. Plastic deformation
iii. Buckle fractures
iv. Green stick fractures
v. Complete fractures

Physeal Injuries

These injuries are also known as Growth plate injuries, Epiphyseal plate injuries and Epiphyseal cartilage injuries.

Note: *These injuries are loosely referred to as epiphyseal injuries. However, it is incorrect because 'physis', 'epiphyseal plate', 'epiphyseal cartilage', refer to the growth plate. Whereas, epiphysis refers to a secondary center of ossification. The growth plate is flanked by epiphysis on one side and metaphysis on the other. Hence, the physeal injuries/growth plate injuries, can extend to the epiphysis or metaphysis or both (Refer classification).*

Structure of a Physis (Fig. 15.1)

The physis consists of logitudinally placed chondrocytes and extracellular matrix. Four zones are identified. From the epiphyseal end to the metaphysis. They are arranged as follows:

- Germinal zone
- Proliferative zone
- Hypertrophic zone
- Zone of endochondral ossification

There is abundant extracellular matrix in the germinal and the proliferative zones. Thus, they are inherently strong and resist shear. The hypertrophic zone has mature uncalcified hypertrophic cells with scanty matrix. Hence, its inherent strength is poor. The zone of endochondral ossification has calicified cells and it blends smoothly with the metaphysis. This increases its inherent stability.

Thus, the weakest area of the physis is the zone of hypertrophic cells through which most of the injuries occur.

Table 15.1

Comparison of pediatric and adult skeleton.

Differences seen	*Pediatric skeleton*	*Adult skeleton*
Growth	Growing, with the presence of epiphysis, physis and metaphysis	Growth is complete. Bone is mature and fully ossified.
Plasticity	Present	Absent
Periosteum	Thick and loosely attached	Thin and firmly attached
Healing	Rapid	Slow and steady
Remodeling capacity	High	Low. Capacity decreases with advancing age
Plastic deformation	Possible	Not possible

Circumferentially, the core of physis is surrounded by an outer perichondrial fibro-chondro-osseous ring, which is sometimes referred to as 'Periphysis'. This ring, is responsible for the stability of the physis. The portion of the periphysis that surrounds physis is known as the 'Zone or Groove of Ranvier'. That portion of this ring which is adjacent to the metaphysis is known as the 'Ring of La Croix'.

The Zone of Ranvier is a wedge shaped invagination consisting of cells contiguous with the epiphysis and is responsible for the latitudinal (widening) growth of the physis.

Epiphyseal arteries enter the physis and the terminal branches end at the proliferative zone.

The diaphyseal nutrient artery with its loop like capillary network, supplies the metaphyseal end of the physis (growth plate).

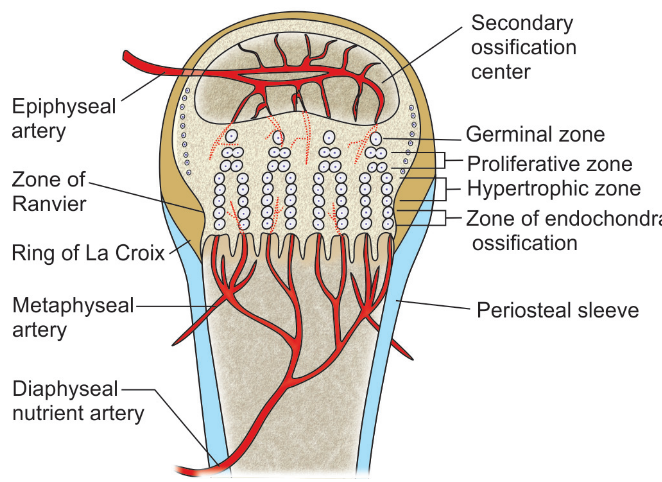


Figure 15.1

Diagrammatic representation of the structure of the physis.

Classification of Physeal Injuries based on Salter and Harris (Fig. 15.2)

Type I: Shearing injury extending through the entire physis (Figs 15.3A and B).

Type II: Shearing injury extending through the portion of the physis and exiting through the metaphysis.

Type III: Shearing injury extending through the portion of the physis and exiting through the epiphysis.

Type IV: Injuries extending from the epiphysis through the physis into the metaphysis.

Type V: Crushing injury to the physis.

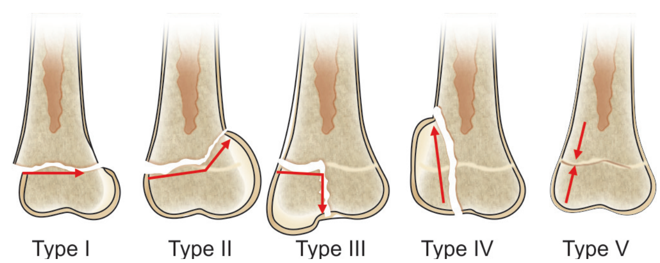
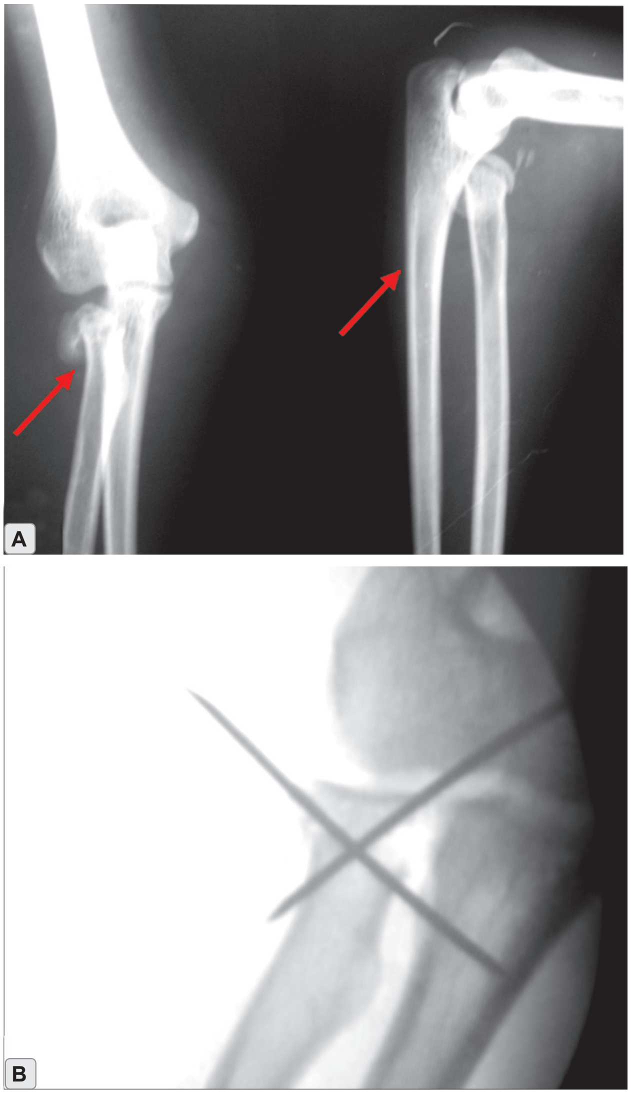


Figure 15.2

Pictorial representation of the physeal injuries based on Salter and Harris classification.

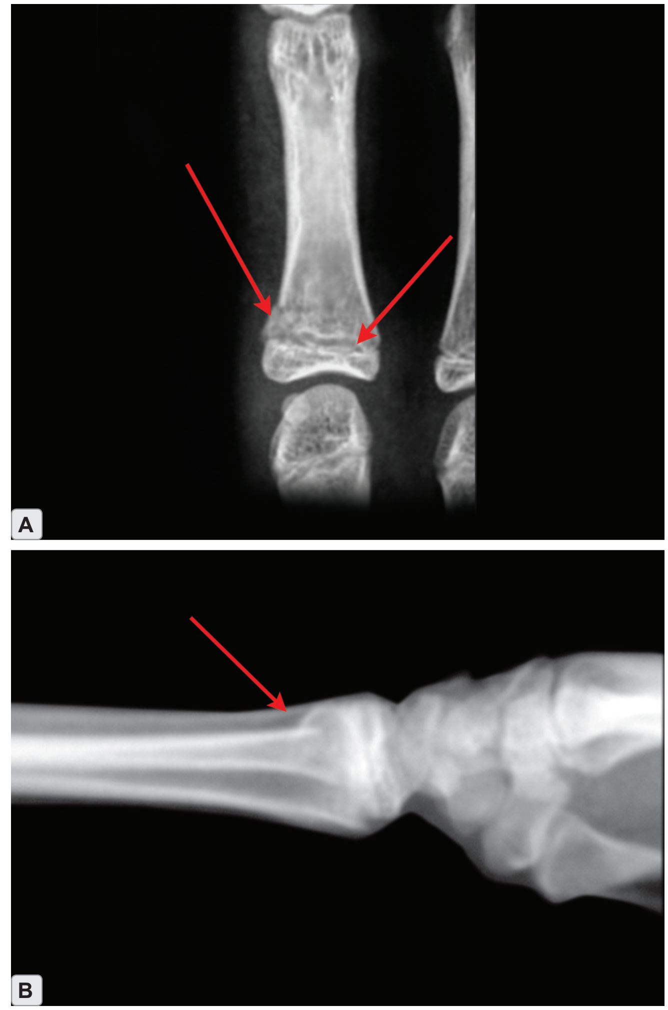
Thurston Holland Sign (Shiny Corner Sign)

This is a sign seen in type II physeal injury. The triangular metaphyseal fragment is the only indicator of this injury (Figs 15.4A and B). This is because, the deforming force would have already had passed through and sheared the rest of the physis, before causing fracture and exiting from the area of triangular metaphyseal fragment. The deformation is not made out as the displaced fragment falls back into position. Hence, when the sign is present, these injuries need attention like any other displaced type II physeal injuries.



Figures 15.3A and B

Type I Salter and Harris injury involving radial epiphysis. (A) Preoperative and (B) Intraoperative.



Figures 15.4A and B

(A) The Salter and Harris type II physeal injury involving the base of the proximal phalanx of the index finger and (B) lower radius. Note the triangular metaphyseal fragment the 'Thurston Holland sign', which is the only indicator of type II physeal injury. When missed, may lead to malunion.

Diagnosis of Physeal Injuries

X-ray: A good radiograph gives the diagnosis in majority of the cases when ossific nucleus of the epiphysis has appeared. If not, when the injury is suspected, one has to resort to MRI.

Management

Accurate management of these injuries is essential to avoid deformities and growth abnormalities.

Aims of management:

- To anatomically reduce the physis.
- To stabilize the reduction obtained either externally by cast or internally by closed pinning/open reduction and internal fixation.

During the above procedures it is very essential to protect the germinal layer and avoid damage to it.

In those cases which present late, always consider secondary corrective procedures.

When the reduction is nonanatomic, following factors are helpful in assessing the outcome and making decisions.

Age of the patient: Younger the age, greater is the remodelling capacity.

Type of the injury: Types I and II have better chance of remodelling than III and IV.

Location of the injury: An injury in the vicinity of a weight-bearing joint is subjected to greater stress than an injury near a nonweight-bearing joint. Hence, a nonanatomic reduction is more acceptable near a non-weight bearing joint.

Duration of injury: More is the lapse of time, poorer is the outcome of primary treatment procedures.

Complications

Inherent complications
- Malunion
- Osteonecrosis
- Growth arrest

Associated complications
- Infections
- Neurovascular injury

Management of inherent complications: Complications of malunion and osteonecrosis are managed by taking into consideration the resultant deformity and disability. Carefully planned corrective osteotomies considerably improve the function.

Growth disturbance: Disturbance develops because of the formation of a bony bridge or a bar across the physis or retardation of growth following injury. After careful assessment of growth retardation and adequate planning, procedures like resection of the bony bar, epiphyseodesis, etc. will help in the correction of the discrepancy.

Management of associated complications: These are managed accordingly like other cases of infection and neurovascular injury.

Harris growth arrest lines:
These when present/absent help in determining nature of the arrest.
***Absence** indicates total physeal injury and no growth has occurred after injury.*
***Transverse and parallel** lines indicate normal growth pattern after an intermittent arrest following physeal injury.*
***Asymmetric** growth line indicates partial injury to the physis.*

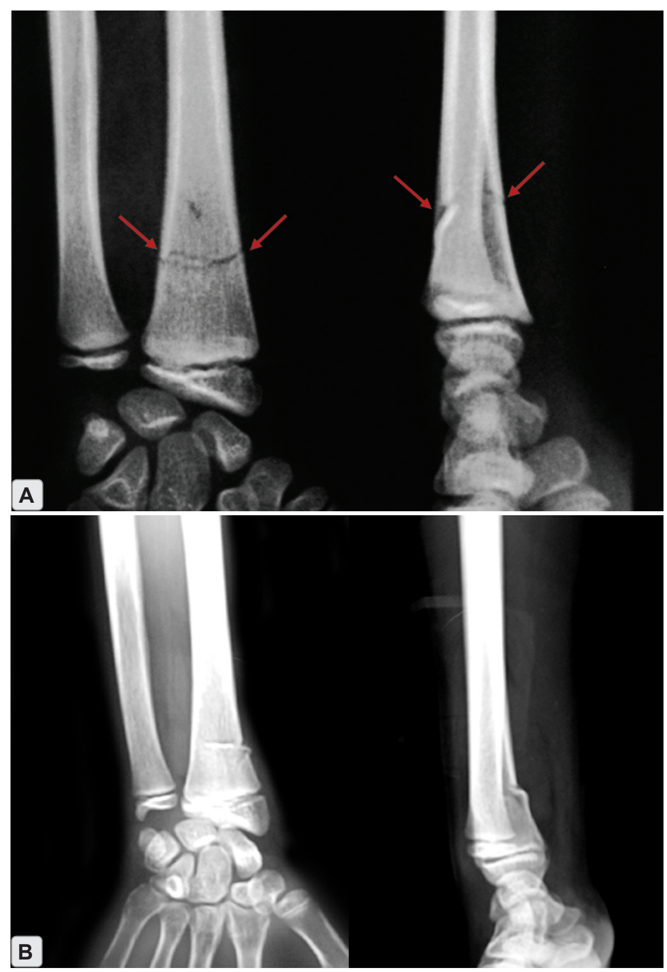
Plastic Deformation

Borden, first described the plastic deformation of bones in children. Immature bone is weaker in bending strength. Hence, it absorbs more energy and bends before the fracture occurs. This results in plastic deformation. Forearm is the common site for plastic deformation and ulna is the common bone involved. Some people have reported cases of the plastic deformation occurring in the femur.

Lesser degrees of deformation get remodelled with growth. But, deformities in the forearm more than 20° with limitation of pronation and supination need correction. Immobilization for a full period of 4–6 weeks is essential for a good healing.

Buckle Fractures (Figs 15.5A and B)

Also known as torus fractures because radiologically a ridge of bone (torus) is observed. These injuries are common at the transitional area between the woven



Figures 15.5A and B

Radiographs of (A) a complete fracture with a buckle (B) incomplete fracture with a buckle. The presence of thick periosteum prevents displacements.

bone of the metaphysis and the lamellar bone of the diaphysis. The fracture may present as:

a. A simple cortical break with a ridge (Fig. 15.5B).
b. A ridge with plastic deformation of the rest of the bone.
c. A ridge with complete break of the rest of the bone (Fig. 15.5A).

A simple break with formation of a ridge can be managed with a well padded splint and it heals uneventfully. More severe forms of break, get displaced over a period of time and result in deformities. To prevent this, they need correction and immobilization in a well-moulded plaster cast.

Greenstick Fractures

These are commonly seen in children (Ref Chapter 1: Fig. 1.9). In these, the fracture is not seen as a complete break though the entire bone is involved. A part of the bone breaks and the rest of the bone bends like a Greenstick. Hence the name. The bone on the tension side breaks completely. The bone on the compression side bends and undergoes plastic deformation because of thick periosteum. It is necessary to break the intact cortex on the compression side to achieve a good reduction. Reduction should be maintained for a full period of approximately 6 weeks to avoid the risk of re-fracture due to premature removal of the plaster cast.

COMMON FRACTURES IN THE LOWER LIMB: DIAGNOSIS AND MANAGEMENT

Fracture of Shaft of Femur in Children

Mechanism of Injury

The trauma should be of a severe nature for this fracture to occur. Fall from a height, motor vehicle accidents are some of the examples of severe trauma.

In the younger age group child abuse must be suspected especially when there is delay in seeking the medical treatment and there are other injuries.

Diagnosis

It is a straight forward diagnosis. X-rays help in assessing the nature of the fracture and planning the treatment.

Treatment

Following factors help to decide the type of treatment to be instituted.

i. Age of the child
ii. The degree of comminution
iii. The degree of displacement, amount of over-riding and shortening
iv. Open wounds with disturbed soft tissue envelop
v. Vascular injury
vi. Other associated injuries

Zero to six months of age: Excellent results are obtained with non-operative treatment. Strapping the thigh to the abdomen in a newborn, using the Pavlik harness in an infant, are some of the methods employed. Compression of the femoral nerve during the treatment is a possibility because of flexed position of the limb and should always be kept in mind.

Six months to two years: Immobilization with

- Bryant's traction (Fig. 15.6).
- Closed reduction and spica cast application.

Two to five years: Skin or skeletal traction on a splint followed by protection in a spica cast or a brace. Spica cast gives a better protection in children for the simple reason that a child does not understand the importance of nonweight-bearing and tries to walk when the brace is given. Also, children tolerate this extensive plaster very well.

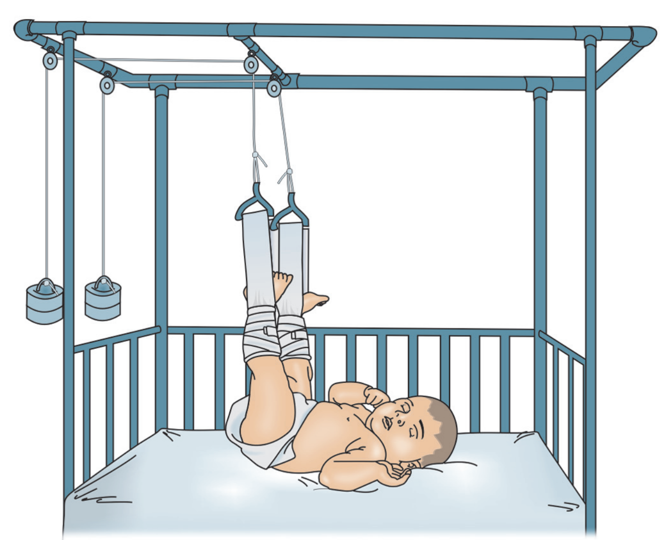


Figure 15.6

Bryant's otherwise known as Gallows traction. Note how the buttocks are lifted off the bed. This is essential for effective traction.

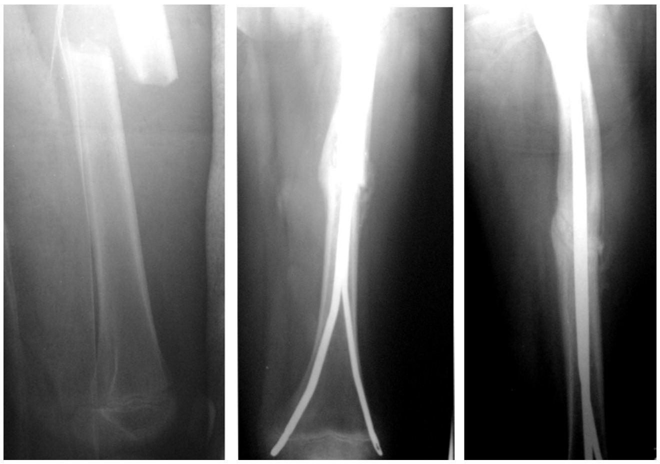


Figure 15.7

Closed reduction and fixation of fracture of shaft of femur using elastic nails.

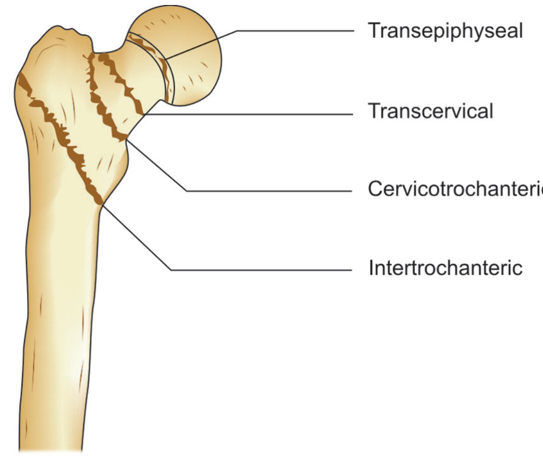


Figure 15.8

Diagram showing different types of fractures of the neck of the femur in children according to Delbet's classification.

Closed reduction and flexible nailing OR open reduction and flexible nailing may be necessary in selected cases, e.g. when shortening is more than 3 cm and not amenable for manipulative correction.

Five to twelve years: Closed/ open reduction and flexible nailing is the treatment of choice (Fig. 15.7) except in those minimally displaced and long oblique fractures which are amenable for closed treatment with traction.

Minimally invasive plate osteosynthesis should be reserved for cases with segmental comminution. Such fractures are best stabilized by bridge plate technique.

Open fractures: External fixators are used in the treatment of open fractures.

Vascular injuries need immediate repair and other associated injuries are dealt with accordingly, e.g. visceral injury, chest injury, head injury, etc.

Fracture of Neck of Femur in Children

Mechanism of Injury

Always occurs as a result of high energy trauma or fall from a height. Associated injuries are common.

Classification

Based on Delbet's classification four types are identified (Fig. 15.8).

- Type I: Transepiphyseal fractures
- Type II: Transcervical fractures
- Type III: Cervicotrochanteric fractures
- Type IV: Intertrochanteric fractures

Diagnosis

X-rays give adequate information of the injury. Diagnosis is straight forward.

An MRI may be necessary to evaluate the vascularity of the femoral epiphysis during the follow up period after treatment. Use of titanium implants allows MRI to be done with implants in situ.

Treatment

Closed/open reduction and internal fixation is the treatment of choice. It is done at earliest, ideally within 24 hours. Hematoma is to be aspirated from the joint.

Bigelow's Dictate: In 1864, Bigelow remarked that "*While the impacted fracture of the base of the femoral*

neck unites by bone, if at all, there seems to be a decreasing tendency to osseous union as we approach the smaller portion of the neck near its head".

The above statement is found to be true even in pediatric femoral neck fractures. Worst prognosis is seen in transepiphyseal fractures and best prognosis is seen in intertrochanteric fractures. The prognosis is found to become better from type II to type III fractures.

FURTHER READING

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2. John A Herring (Ed). Tachdjian's Pediatric Orthopedics. 4th edition, Saunders: Elsevier, Philadelphia. 2008;3.
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16 Poliomyelitis

- *General information*
- *Epidemiology*
- *Pathogenesis*
- *Treatment of poliomyelitis*
- *Immunization and its current status*
- *Skeletal deformities in poliomyelitis*
- *Deformities around the hip*
- *Deformities of foot and ankle*

GENERAL INFORMATION

Jakob Heine in 1840, first recognized Poliomyelitis as a distinct entity. In 1988, the World Health Organization, together with Rotary International, UNICEF, and the US Center for Disease Control and Prevention passed the Global Polio Eradication Initiative, with the goal of eradicating polio by the year 2000. On 20th August 1994 the Americas were certified as polio-free. This was achieved by the help of oral polio vaccination. The oral polio vaccine must be kept at 2–8° celsius for vaccination to be successful.

EPIDEMIOLOGY

Poliomyelitis is a viral disease caused by a human enterovirus belonging to the family of Picornaviridae and the genus human enterovirus C. Structurally, it is similar to human coxsackie, echovirus and rhinovirus. It was first isolated in 1909 by Karl Landsteiner and Erwin Popper. It is composed of a RNA genome and a protein capsid and is considered as one of the most well characterized virus. Three different strains PV1, PV2 and PV3 are identified, the variation being in the protein capsid. All three strains are highly infective and PV1 is the most common strain that infects, among the three.

PATHOGENESIS

The route of spread is fecal-oral and once the virus is ingested, the replication immediately occurs in the gastrointestinal tract because of following reasons.

a. It can survive in the highly acidic medium of the gastrointestinal tract.
b. It can replicate quickly even before the immune response is generated.

The initial spread occurs through the lymphatic system throughout the body tissues. The primary viremia which occurs is only transient. When sustained replication begins, secondary viremia occurs. This manifests with symptoms like fever, headache, sore throat, etc. During secondary viremia, virus may enter the central nervous system (CNS) through the bloodstream and the nerves.

When the virus enters the central nervous system, the motor neurons in the spinal cord, brain stem and motor cortex are affected and paralysis occurs. Paralysis is proportionate to the site and type of neurons involved.

Some of the neurons temporarily lose their function during viremia and then recover fast while other neurons die causing permanent loss of function. The paralysis seen is asymmetrical and lasts for a few weeks after the acute stage before the recovery begins. Maximum recovery occurs within 6 months and proceeds up to 24 months. Residual paralysis which remains after 24 months is permanent.

Extent of residual paralysis is directly proportional to the dead and damaged motor neurons.

Mode of spread to CNS: The mode of spread is not clear and three hypotheses have been put forth.

a. The virions directly cross the blood brain barrier and enter the CNS.
b. Retrograde axonal spread through soft tissues bathed with viremic blood.
c. Entry via infected monocytes and macrophages.

Mechanism of entry into the host cell: After binding with the host cell to immunoglobulin like receptor CD 155, entry into the cell can occur by

a. Formation of a pore in the plasma membrane.
b. Receptor mediated endocytosis.

Nature and Result of Paralysis

- Involvement of motor cortex and bulbar motor neurons—results in respiratory paralysis and death.
- Involvement of spinal cord neurons—results in paralysis of muscles of trunk and limbs.

Stages of Poliomyelitis

Three distinct stages are recognized.

a. Acute stage: This stage is from the onset of symptoms to establishment of paralysis. Lasts for about 14–16 weeks.
b. Stage of recovery or convalescence: This stage occurs from establishment of paralysis to maximum recovery. Begins at 14–16 weeks and lasts up to 24 months.
c. Stage of residual paralysis: This establishes after 24 months when no recovery is possible.

Differential Diagnosis

The onset mimics any of the viral fever such as influenza, or bacterial infection. Once the paralysis occurs, conditions like Guillian-Barre syndrome, Postdiphtheritic paralysis, meningitis, etc. should be considered. But the striking feature in poliomyelitis is asymmetrical paralysis.

Laboratory Diagnosis of Poliomyelitis

1. Viral Isolation: Poliovirus may be recovered from the stool or the swab from the pharynx of a person with presumed poliomyelitis. Isolation of virus from the cerebrospinal fluid (CSF), collected by lumbar puncture is diagnostic, but is rarely accomplished.

   Note: *If at all poliovirus is isolated from a person with acute flaccid paralysis, it must be tested further using oligonucleotide mapping or genomic sequencing, to determine whether the viral strain is "wild-like" or "vaccine-like." It is very important to determine the source of the virus because for each reported case of paralytic polio caused by wild poliovirus, it is estimated that another 200 to 3,000 contagious asymptomatic carriers exist in the community.*

2. CSF analysis: The CSF in poliovirus infection generally contains an increased number of white blood cells (10 to 200 cells/mm, primarily lymphocytes) and a mildly elevated protein from 40 to 50 mg/100 ml.
3. Serology: Antibodies to poliovirus can be diagnostic, and are generally detected in the blood of infected patients, early in the course of infection. But, neutralizing antibodies too appear very early and may be at a high level, by the time the patient is hospitalized. Therefore, a four-fold rise in antibodies may not be demonstrated.

Treatment of Poliomyelitis

In the acute stage: The treatment is only symptomatic. Aims at reducing the temperature, generalized body ache and muscle pain.

In the recovery or convalescence phase: Active and expert team work with physiotherapeutic intervention is necessary to prevent deformities and facilitate recovery from paralysis. Braces, splints and plaster casts are indicated to accomplish this.

In the stage of residual paralysis: Surgical procedures are indicated for improving the function of paralyzed part and reduce the disability.

IMMUNIZATION AND ITS CURRENT STATUS

The World Health Organization together with Rotary International, UNICEF and the US Center for Disease Control and Prevention, planned a global polio eradication initiative in the year 1988. Because of this, successful eradication of the disease was possible in many countries and the American continents were certified as disease free in the year 1994. Peru reported the last case in the year 1991.

For a vaccine to be successful the cold chain of the preparation of live attenuated virus should be maintained at 2–8° celsius. In many of the endemic areas basic health infrastructure is lacking. Further in tropical countries where the outside temperature is too high, effective vaccination becomes difficult.

Even among those individuals who receive oral polio vaccine, only 95% will develop immunity. Which means that 5 out of 100 individuals vaccinated will not develop any immunity and are susceptible to developing poliomyelitis. Further it is a well-known fact that the poliovirus is transmitted only through contact from person-to-person, which means that the transmission cycle of polio is from one infected person to another normal person who lacks immunity and is susceptible to the disease.

So when there is a susceptible population of 5% even after vaccination, it is difficult to achieve eradication unless the herd immunity increases. Only herd immunity can protect these individuals and as per the concepts of herd immunity this can be achieved only if the herd immunity is high in the range of 80–86%. For the same reason, it is not possible to stop routine immunization campaign as it would increase the population of nonimmunized individuals which in turn increases the susceptibility by decreasing the percentage of herd immunity.

Further in 21st century two new challenges have been observed.

1. *Unobserved polio transmission:* In some individuals the symptoms of poilomyelitis are mild and they are unaware that they are afflicted with the disease. But when susceptible individuals come into contact with these patients they develop a severe form of poliomyelitis. This explains sudden onset of widespread epidemic before detection of any cases.
2. *Mutation:* The mutations or recombinations developing in the attenuated strain used in the vaccine (OPV) also can lead to a sudden outbreak of the disease. The term Circulating Vaccine Derived Polioviruses is used to identify such strains (cVDPVs). Because of these strains currently there is a need for more expensive inactivated polio vaccine (IPV) in order to eradicate the disease. These are given as injections.

Immunization schedules: Following recommended schedules of polio vaccination for children are practiced currently.

a. Two doses of IPV at 2 and 4 months of age, followed by two doses of OPV at 12–18 months and 4–6 years.
b. All IPV: IPV can be given at 2, 4, and 12–18 months, and 4–6 years.
c. All OPV: OPV can be given at 2, 4, and 6–18 months, and 4–6 years.

SKELETAL DEFORMITIES IN POLIOMYELITIS

In majority of the cases, the deformities are seen in lower limbs. Trunk and upper limbs are less frequently affected. These deformities can be prevented to some extent by judicious use of braces, plaster casts and splints during the phase of recovery. Repeated evaluation is necessary to prevent and minimize the residual deformity. Gross muscle imbalance and altered growth pattern are the ones which are resistant for non-operative correction and need well-planned surgical procedures for correction of deformity.

Pathogenesis of Deformities

The skeletal deformities in poliomyelitis occur due to:

a. Muscle imbalance
b. Severe sustained muscle spasm
c. Faulty posture
d. Gravity
e. Dynamics of activity
f. Altered growth pattern after recovery

Effect of muscle imbalance: Paralysis of a muscle in the presence of healthy antagonistic muscles allows the joint to remain fixed in one position and over a period

of time this results in fixed deformity, e.g. an equinus deformity developing after paralysis of ankle and foot dorsiflexors. The intact triceps surae (gastrosoleus) which is a plantar flexor assisted by toe flexors, pulls the ankle and foot into plantar flexion. If not identified, over a period of time, an equinus deformity develops.

Effect of gravity: A paralyzed limb may adopt a position dictated by gravity and remain so for quite sometime unattended. This results in soft tissue contracture and a fixed deformity. The commonest example is development of a flexion, abduction deformity at the hip.

Effect of dynamics of activity: Weight bearing stress has a deforming effect on the paralyzed joint. Because of lack of functioning muscles and tendons, the joint is unable to stabilize itself against these weight bearing stresses and tends to get deformed. The ligaments and the capsule become lax due to over stretching and ultimately the relation between the two bones at the articulation gets disturbed.

Altered growth pattern after recovery: When the muscles remain paralyzed for a long time due to inactivity, there is no stimulus for the limb to promote growth. The blood supply to the bone derived through the muscles, also becomes deficient. This results in altered growth pattern and asymmetric growth of the skeleton and limb length discrepancy.

Preoperative Considerations

Surgical correction is rarely indicated during the convalescent stage of poliomyelitis which lasts for nearly 24 months, after the period of acute illness. It is in the residual stage which lasts throughout the life that the orthopedic procedures are indicated.

Surgical procedures are indicated only in a patient whose physical handicap decreases after the procedure. If a person has adapted himself naturally to the deformity and is carrying out his routine without much difficulty, surgical correction is not indicated.

Every patient with poliomyelitis is to be considered and evaluated as an individual and a carefully planned rehabilitation schedule is to be outlined. Evaluation of trunk and upper limbs is absolutely essential in a patient with lower limb deformities, as they play a great role in ambulation after correction of deformities.

Design of Surgical Procedures

These procedures aim at preventing and correcting the deformities and abolishing the need for wearing external supports like braces, splints and corsets thus making the patient as independent as possible. When needed, the use of crutches and canes for ambulation is considered acceptable.

The surgical procedures for deformity correction are basically designed to balance the muscle power, stabilize the flail joints and to correct the limb length discrepancy. They are classified as soft tissue procedures and bony procedures.

The soft tissue procedures

a. Release of contractures
b. Tendon transfers

The bony procedures

a. Arthrodesis
b. Osteotomies
c. Limb lengthening procedures.

When bony procedures are planned in children, care must be taken that they do not severely disturb the growth in future. Also, the correction can be planned in one stage or in multiple stages. It is true that early correction gives better results. Older patients with severe, longstanding deformities pose technical challenges for the operating surgeon. Also, the rate of complication is high in such cases.

Finally, a surgeon should not hesitate to advise a wheel chair for a severely paralyzed patient than venture into surgery and give cumbersome braces and crutches for mobilization.

DEFORMITIES AROUND THE HIP

Deformities around the hip are likely to affect weight bearing and ambulation. They occur mainly due to muscle imbalance and effect of gravity. When the contracture is severe and bilateral, patient adapts quadruped locomotion. Only after release of contractures, upright locomotion is possible. Different deformities seen in the hip region are summarized in Table 16.1.

Table 16.1

Various deformities around the hip and the muscle imbalance responsible for the same.

Deformity at the hip	*Muscle(s) affected (weak or paralyzed)*	*Muscle(s) intact and acting (unopposed)*
Flexion	Gluteus maximus	Iliopsoas
Flexion, abduction, External rotation (common)	All the glutei, the adductor group and the internal rotators	Iliopsoas and the tensor fascia lata
Flexion, adduction, internal rotation (rare)	All the glutei and the external rotators	Iliopsoas and the adductors
Paralytic dislocation of the hip (polio before 2 years of age)	Glutei	Flexors and adductors

Role of Iliotibial Band in Contractures

Fascia lata is attached proximally to coccyx, sacrum, iliac crest, inguinal ligament and arch of the pubis. Distally, it is attached to all the bony prominences over the femoral and the tibial condyles, the head of the fibula and the patella. Through the intermuscular septae, it not only invests all the gluteal and the thigh muscles but also gives attachment to them.

The iliotibial band is the thickened part of fascia lata which is formed by the convergence of the divisions of fascia lata and extends on the lateral side of the thigh. It is attached throughout the length of the femur to the linea aspera through lateral intermuscular septum.

The plane of the band is anterior and lateral to the axis of the hip and posterior and lateral to the axis of the knee. Hence contracture of either strong muscles (biceps femoris, gluteus maximus, vastus lateralis and tensor fasciae latae) or the contracture of the band itself can cause severe deformities across the two joints as well as in the trunk and pelvis as follows.

Around the trunk

a. Pelvic obliquity (secondary to flexion abduction contracture at the hip).
b. Increased lumbar lordosis (secondary to flexion contracture at the hip).

Deformities caused by tight iliotibial band may be grouped as follows.

Around the hip

a. Flexion abduction contracture

Around the knee

a. Genu valgum
b. Flexion contracture
c. External femoral torsion
d. External tibial torsion

Other deformities

a. Secondary talipes equinovarus
b. Limb length discrepancy

Paralytic Dislocation of the Hip

The paralytic dislocation of the hip develops because of muscle imbalance. If a child contracts polio before the age of 2 years and develops paralysis of the gluteal muscles with intact flexors and adductors, paralytic dislocation is certain because of their unopposed action.

Surgical Procedures for Release of Contractures

a. Soutter's procedure: The iliotibial band and the muscles are released from the iliac crest.
b. Yount's procedure: The iliotibial band and fascia lata are divided above the femoral condyle from midline anteriorly to Biceps tendon posteriorly.
c. Transfer of the crest of the ilium (Campbell's): This is indicated in severe deformities. It comprises of releasing the iliotibial band and muscles from the iliac crest, extensively up to the acetabulum, freeing the abdominal muscles from the iliac crest and then resection of a portion of iliac crest.

Surgical Procedures for Paralytic Dislocation of the Hip

Surgical procedures for paralytic dislocation are indicated when the nonoperative method of closed reduction and maintenance of reduction fails.

Before adaptive skeletal changes have taken place, the procedures designed for correction, aim at restoring the muscle balance. After the secondary adaptive changes have taken place, surgical procedures designed, aim at offering stability at the hip which is in addition to muscle transfer.

a. *Mustard's and Sharrad's (modification of Mustard's) tendon transfer:* In this, the Iliopsoas is detached from its insertion to lesser trochanter and transferred to the greater trochanter.
b. *Varus femoral osteotomy:* This is done for secondary valgus of the femoral neck.
c. *Salter's innominate osteotomy and Pemberton's pericapsular osteotomy:* These are done when acetabulum is shallow and there is increase in obliquity of the acetabular roof.
d. *Arthrodesis:* This is done when reconstructive procedures have failed or the hip develops arthritic changes. Preferably it is done in adults.

DEFORMITIES OF FOOT AND ANKLE

Like every deformity in the lower limb, foot and ankle deformities also are subjected to dynamics of weight-bearing and effect of gravity. They progress and become fixed over a period of time, if unattended. To begin with, these deformities are flexible. But, if neglected, structural changes develop in the bones and joints and the deformities tend to become rigid as the skeleton matures. Still if they remain unattended, degenerative changes set in and the foot and ankle become painful.

Since ankle and foot plays a major role in weight bearing, it is important to prevent these deformities whenever possible or if already developed to correct them with appropriate surgical procedures at earliest.

To understand the pathogenesis of various deformities, it is important to remember the fact that every deformity in poliomyelitis begins with muscle imbalance, which means, if one group of muscles is paralyzed the part is subjected to the forces generated by the opposite group. Thus, the balancing effect of two opposite groups of muscles is lost and a deformity develops.

Considering this fact, **Peabody** has precisely classified the muscle imbalances of foot and ankle. According to Peabody the imbalances that develop is classified as follows:

a. Limited extensor-invertor insufficiency
b. Gross extensor-invertor insufficiency
c. Evertor insufficiency
d. Triceps surae insufficiency

In younger age group, before structural changes develop, appropriate tendon transfers are useful in correcting the imbalance, prevention of deformity and stabilizing the foot and ankle. To understand the concepts of tendon transfer and for achieving good results, the age of the patient should be atleast 10 years and more.

Requisites for a sucessful tendon transfer: Following are the parameters to be considered while planning a tendon transfer.

1. Agonists are preferred to antagonists (helps in muscle re-education after transfer).
2. Power should be minimum of Grade-4 (after transfer the muscle loses power by one Grade).
3. Should have almost similar range of excursion to that of paralyzed tendon.
4. Joint across which the tendon acts should be supple without limitation of range of movement.
5. The nerve supply and the blood supply should not get impaired during the procedure.
6. The transfered tendon should glide in its own sheath, sheath of an adjacent tendon or in the subcutaneous fat.
7. The insertion should be in line and close to the insertion of the paralyzed tendon.
8. Secure attachment with increased tension is desirable.

Tendon Transfers and Corrective Procedures for Muscle Insufficiency

a. *Limited extensor-invertor insufficiency:* In this, only tibialis anterior is paralyzed. This results in a progressive deformity of equinus and valgus sometimes an associated cavus.
   Equinus—corrected by stretching or Tendo-Achilles lengthening. This is followed by transfer of the extensor hallucis longus to the base of the 1st metatarsal.
   Cavus—Corrected by plantar fasciotomy.
   Valgus—Gets corrected with the transfers.
   If the deformity is fixed and the foot is skeletally mature, triple arthrodesis is necessary along with tendon transfer.
b. *Gross extensor-invertor insufficiency:* In the less severe form, only tibialis anterior, extensor hallucis longus (EHL) and extensor digitorum longus (EDL) are parlayzed. In severe form both tibialis—anterior and posterior, and the extensors of the toes (EHL, EDL and EDB) are paralyzed. Restoration is done as follows:
   In less severe form—the peroneus longus is transferred to the dorsum of the foot and inserted medially into the medial cuneiform or more laterally into the 4th metatarsal depending on the power of tibialis posterior (for balancing).
   In more severe form—transfer of both peroneus longus and brevis to the dorsum of the foot is necessary for correcting the deformity.
   Triple arthrodesis is also indicated along with tendon transfer when there are structural changes.
c. *Evertor insufficiency:* This is the result of varying grades of paralysis of peroneal muscles. The foot goes into varus deformity.
   In mild form—varus is corrected by shifting the extensor hallucis longus to the base of the 5th metatarsal.

In severe form—split tibialis anterior transfer with the lateral half securely fixed to the insertion of the peroneus brevis tendon or transfer of extensor hallucis to the base of the 1st matatarsal and tibialis anterior laterally to the cuboid bone are the procedures that are commonly done.

d. *Triceps surae insufficiency:* Paralysis of triceps surae results in calcaneus deformity with an element of either valgus or varus. The other deformity that can develop is calcaneocavus.

Early correction is indicated for this insufficiency.

Calcaneus—corrected by transfer of tibialis anterior to the calcaneum and extensor hallucis longus to the base of the 1st metatarsal.

Calcaneovalgus—transfer of peroneal tendons to the calcaneus.

Calcaneovarus—transfer of tibialis posterior and the flexor hallucis longus to the calcaneus.

Calcaneocavus—transfer of both peroneal tendons and tibialis posterior to the calcaneus.

Triple arthrodesis is indicated in long standing deformity for correction of distorted anatomy.

Bony Procedures for Deformity Correction

While tendon transfers balance, the muscle imbalance, provide the necessary motor power for substituting the function of the paralyzed muscle and give dynamic stablity, the bony procedures fix the joints, stabilize the foot and give static stability. Triple arthrodesis is one among such procedures which is commonly done to give static stability to the foot.

Triple Arthrodesis

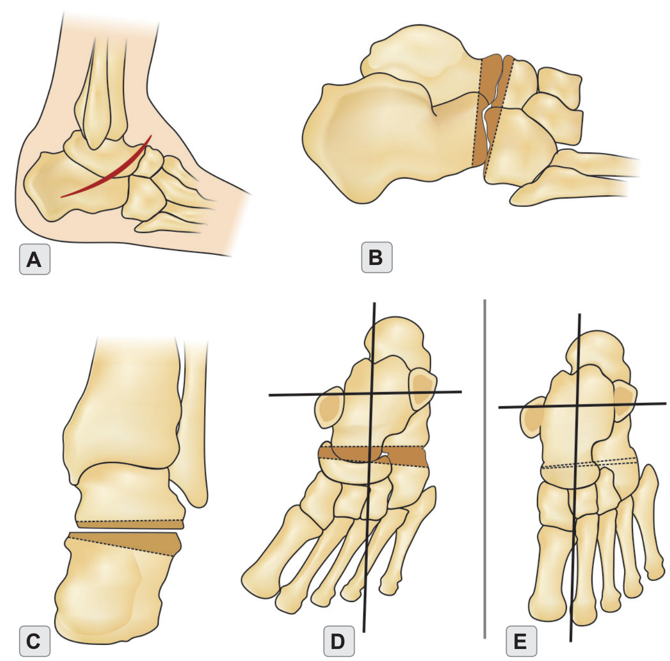
As the name itself suggests three joints are fused in this procedure. The procedure shortens the foot a little. The three joints that are fused are:

a. The subtalar joint
b. The talonavicular joint
c. The calcaneocuboid joint

When triple arthrodesis is combined with tendon transfers, it helps in minimizing the joints across which the transferred tendon should act and improves the weight-bearing stability. To avoid retardation of growth, triple arthrodesis is always done after skeletal maturity. More so, in poliomyelitis, where the bone growth is already retarded due to poor musculature, paralysis of muscles and small foot. The two different types of triple arthrodesis are shown in Figures 16.1 and 16.2.

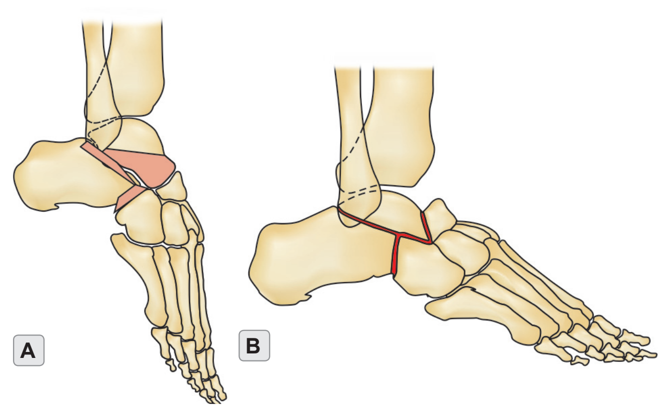
Other Bony Procedures

Several other bony procedures are also described for correction of various deformities of the foot, e.g. Hoke's arthrodesis, Dunn's arthrodesis, Siffert, Foster and Nachammie procedure, 'V' tarsal osteotomy of Japas, Anterior tarsal wedge osteotomy of Coles, etc.



Figures 16.1A to E

Ryerson's, classical triple arthrodesis. (A) Classical skin incision for exposure. Appropriate wedges of bone taken at the three joints namely (C) The subtalar, (B and D) Talonavicular and the calcaneocuboid. (E) Correction achieved after closure.



Figrues 16.2A and B

Lambrinudi's triple arthrodesis—in this procedure, the beak shaped talus is inserted into a trough made in the navicular in order to lift up the fore foot which is in equinus. The subtalar and the calcaneocuboid joints are also arthrodesed.

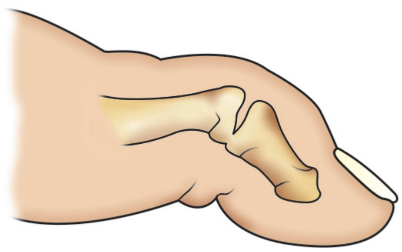


Figure 16.3

The classical hyperextension at the metatarsophalangeal joint and flexion at the interphalangeal joint of the great toe seen in claw toe.

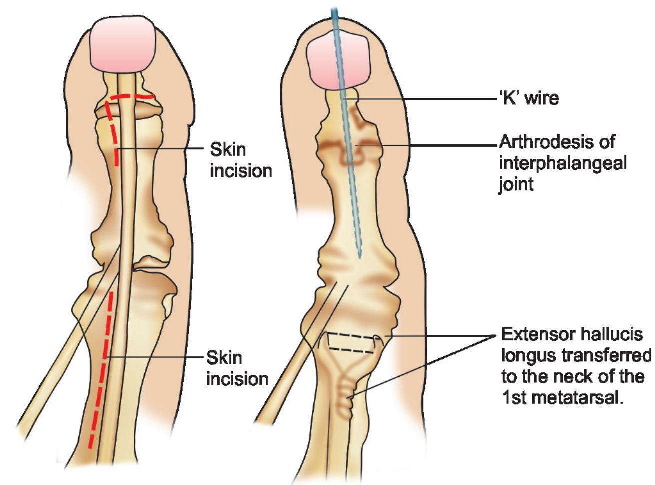


Figure 16.4

The preoperative picture and the picture after modified Jones procedure. The interphalangeal joint is arthrodesed and fixed with a 'K' wire.The extensor hallucis longus is attached to the base of the 1st metatarsal.

Claw Toes

Claw toe is a deformity characterized by hyperextension of the toe at the metatarsophalangeal joint and flexion at the interphalangeal joints (Fig. 16.3). This can occur in polio in two situations.

a. *During push off of stance phase:* In this, it occurs because of weakness of triceps surae. When the long toe flexors try to compensate and assist triceps surae in plantar flexing the ankle for an effective push off, the toes go into flexion at the interphalangeal joints and hyperextension at the metatarsophalangeal joints.
b. *During swing phase:* In this, it occurs because of weakness of dorsiflexors of the ankle. When the long toe extensors assist ankle dorsiflexors to bring about dorsiflexion in order to initiate the swing, the toes go into hyperextension at the metatarsophalangeal joints and flexion at the interphalangeal joints.

Correction of Claw Toes

Great toe: In great toe, the correction of clawing is achieved by means of modified Jones procedure in which the interphalangeal joint is arthrodesed and the tendon of extensor hallucis is divided at its insertion and attached to the neck of the 1st metatarsal (Fig. 16.4).

Other toes: In other toes, correction of clawing is achieved by capsulotomy at the metatarsophalangeal joint and 'Z' lengthening of the long toe extensors or by shifting the insertion of long toe flexors proximally to the bases of the proximal phalanges.

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17 Cerebral Palsy

- *Etiology*
- *Terminologies used in the assessment of limb involvement*
- *Clinical evaluation of a child with cerebral palsy*
- *Treatment*

Cerebral means from the brain. Palsy means paralysis. Hence, the word cerebral palsy means, paralysis as a result of abnormalities occurring in the brain. Depending on the area of involvement it manifests in different ways. Primarily, four areas of involvement are identified.

a. Cerebrum (cerebral cortex)—results in spastic paralysis.
b. Midbrain or base of the brain—results in dyskinesia.
c. Cerebellum—results in ataxia.
d. Extensive involvement—results in rigidity and mixed features.

Right from conception to old age brain can get damaged by a variety of causes resulting in cerebral palsy.

ETIOLOGY

Prenatal Causes

Mainly maternal factors are responsible for prenatal palsy.

a. German measles (rubella) and other viral infections in mother
b. Foetal anoxia
c. Alcohol and drug dependency
d. Genetically determined (consanguinity and conception)
e. Metabolic and endocrinal abnormality, e.g. diabetes mellitus, hypothyroidism, etc.

Natal Causes

Damaging events operating during birth are responsible for natal palsy.

a. Premature delivery
b. Trauma occurring during labor
c. Anoxia occurring during labor
d. Maternal eclampsia

Postnatal Causes

Anoxia and damage occurring to the brain after normal birth is responsible for postnatal palsy.

a. Encephalitis, Meningitis, etc.
b. Trauma, e.g. road traffic accidents, child abuse, etc.
c. Cerebrovascular accident

TERMINOLOGIES USED IN THE ASSESSMENT OF LIMB INVOLVEMENT

Different types of involvement are given different names for the sake of identification of the nature of paralysis and assessment.

Monoplegia

Only one limb is involved. The condition is rare. Other normal extremities need careful evaluation before declaring an individual as monoplegic.

Hemiplegia

The paralysis is generally spastic. Both the limbs on one side are involved. The upper limb is more involved than the lower limb.

Paraplegia

Both the lower limbs are symmetrically affected. They show spastic paralysis.

Triplegia

Three of the four extremities are involved. The condition is uncommon. Careful examination of the normal limb is essential before declaring one as triplegic.

Quadriplegia

All the four limbs are involved. They are subclassified as:

Diplegia: Characterized by involvement of all the four limbs but lower limbs are more involved than the upper limbs.
Double hemiplegia: Characterized by involvement of all the four limbs but upper limbs are more involved than the lower limbs. The pattern is not very common.
Tetraplegia: All the four extremities are equally involved.

Whole Body Involvement

Along with limbs, the trunk, head and neck are also affected.

CLINICAL EVALUATION OF A CHILD WITH CEREBRAL PALSY

It is necessary to evaluate a cerebral palsied child early to have an idea with respect to prognosis. Orthopedically, one is concerned with the child's ability to ambulate.

Signs of Prognosis

Ability to adopt a sitting posture by the age of two years is considered as an indicator of the child being able to walk eventually. Hence, it is a favorable sign.

Signs of Poor Prognosis

The following reflexes when present (except parachute reaction) are poor prognostic indicators for walking.

1. *Asymmetric tonic neck reflex:* When head and neck are turned to one side with the baby in supine position, the arm and the knee on the contralateral side go into flexion. Persistence of this reflex beyond 6 months indicates a poor prognosis (Fig. 17.1).
2. *Moro reflex:* Sudden extension of the neck results in outward movement of the upper limbs followed by slow inward movement towards the chest as if in embracing. A loud noise or sudden jerk may also result in a simliar reaction (Figs 17.2A and B). Persistence of this reflex beyond 4 to 5 months indicates brain damage (Cerebral palsy).
3. *Extensor thrust reflex:* When the child is held upright by the arm pits and lifted up with the feet

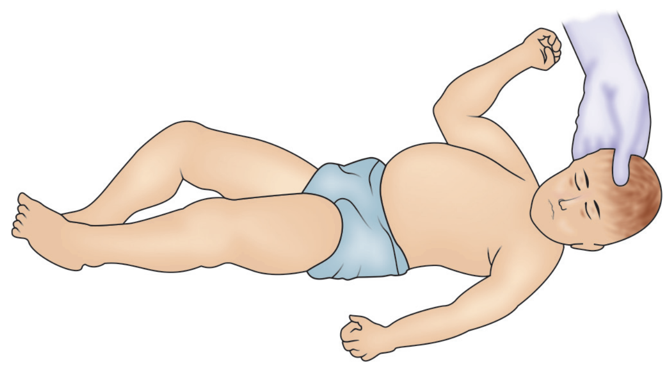
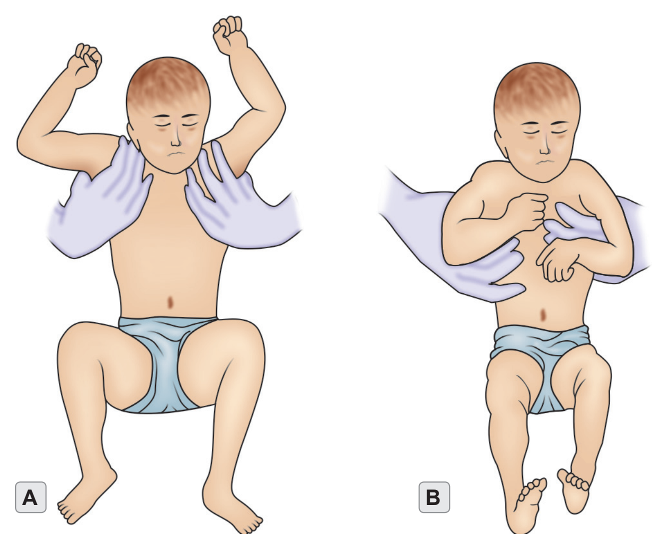


Figure 17.1
Asymmetric tonic neck reflex showing flexion of contralateral upper and lower limb.



Figures 17.2A and B
Moro reflex showing the outward movement of the upper limb followed immediately by inward movement when neck is suddenly extended.

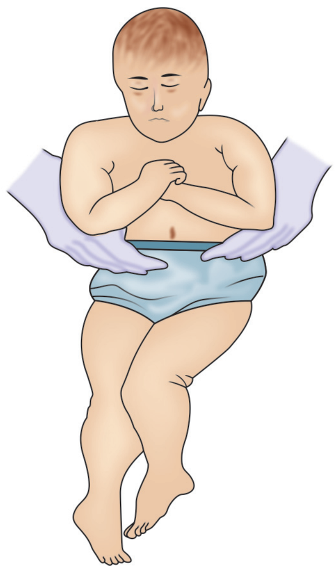


Figure 17.3
Extensor thrust reflex showing spastic stiffening of lower limbs.

Figure 17.4
Neck righting reflex showing the upperlimb, trunk and lower limb following the turned head.

touching the ground (upward pressure on the sole), the lower limbs stiffen out spastically (Fig. 17.3). Persistence of this reflex beyond 2 months, indicates poor prognosis.

4. *Neck righting reflex:* When the head is turned to one side, the shoulder, trunk, pelvis and lower extremities follow the turned head (Fig. 17.4). Persistent reflex beyond 4 months, indicates poor prognosis.

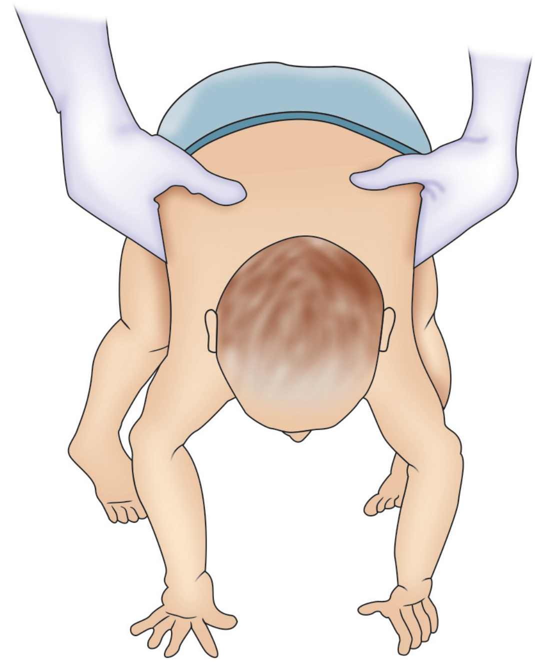


Figure 17.5
Parachute reaction showing protective lowering of the arm in response to lifting. This is normal. Absence of this reflex is abnormal.

5. *Absence of parachute reaction:* Normally, when the child is lifted by the trunk and then lowered forwards to the table, the hands extend towards the table in a protective manner. This is called parachute reaction (Fig. 17.5). This starts developing around 6 months of age. In a child with cerebral palsy this parachute reaction is asymmetrical/absent.

TREATMENT

Cerebral palsy cannot be cured. The deficits which developed as a result of insult to the brain, are permanent. So, the aim of treatment is to improve the functional ability by vocational training. Hence, the methodology adopted has following aims.

1. To increase the emotional maturity.
2. To improve the cognitive abilities such as speech and communication.
3. To enhance the physical independence.
4. To achieve self-sustenance as a final goal.

The assessment of mental capacity is crucial before planning the vocational rehabilitation. The mental capacity determines the functional ability as well the line of training to be adopted.

Role of Surgery

Surgery has a limited role to play in cerebral palsy. It is done mainly in cases of spastic paralysis. Whenever feasible it aims at:

1. Making a bedridden patient sit which in turn helps in improving hygiene and nursing care.
2. Helping a wheel chair bound patient to ambulate.
3. Correcting the deformity in an ambulatory patient thereby increasing his/her functional capacity.

Procedures

A variety of surgical procedures are described both for upper limbs and lower limbs. Basically, they fall into following categories.

1. Surgeries on muscles tendons and other soft tisues. These are designed in the form of:
   a. Release of contractures, e.g. tenotomies and fasciotomies.
   b. Lengthening of tendons, e.g. 'Z' plasty procedures.
   c. Tendon transfers.
2. Neurectomies, e.g. obturator neurectomy (Rarely done these days).
3. Osteotomies and arthrodesis, e.g. Dwyer's closed wedge osteotomy for varus heel, extra-articular subtalar arthrodesis, etc.

These surgeries can be done alone or in combination depending on the need.

18 Gait

- *Definition*
- *Phases*
- *Gait cycle*
- *Abnormal gaits*
- *Trendelenburg gait*

DEFINITION

A normal gait is defined as a forward propulsion of the human body occurring as a result of rhythmic, sequential movements taking place in the limbs (both upper and lower) maintaining the center of gravity and with minimal expenditure of energy.

Upper limbs in humans sway during the gait cycle to maintain the center of gravity.

PHASES (FIG. 18.1)

A. Stance phase
   Which is further divided into:
   - Heel strike
   - Foot flat
   - Mid stance
   - Heel off
   - Toes off (push off)

B. Swing phase
   Which is further divided into:
   - Acceleration
   - Mid swing
   - Deceleration

It is the alternating pattern of movements taking place between the stance phase of one limb and the swing phase of the other that makes propulsion (walking) possible.

GAIT CYCLE

Gait cycle is defined as the cyclical movement taking place from heel strike of one limb to its next heel strike.

Thus to conclude, for a normal gait a healthy support, i.e. the bone; a healthy power, i.e. the muscle and a healthy stimulus to initiate the action, i.e. the intact nervous system is necessary. If any of these are affected it leads to abnormal gait.

Common Terminologies Used in the Analysis of Gait

- *Step length*: The distance between heel strike of one foot and the heel strike of the other foot.
- *Stride width*: Side to side distance between the two feet.
- *Stride length*: The distance between heel strike of one foot to the subsequent heel strike of the same foot. In a normal gait, it is double the step length.
- *Cadence*: Number of steps taken per minute is known as cadence and recorded as steps/min.
- *Velocity*: It is the distance covered in a unit of time. Recorded as meters/min.
  Calculated by Cadence × Step length.
  Velocity varies during gait (walking).
- *Comfortable walking speed (CWS)*: It is walking with least energy consumption during a unit of time. Normally expressed as meters/min. With increase

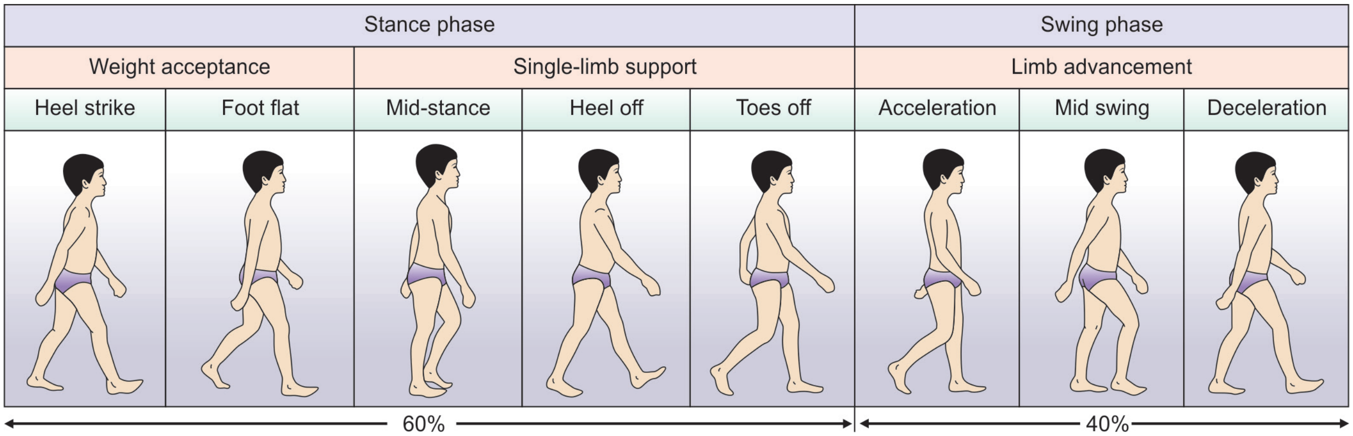


Figure 18.1

Various phases of gait and weight distribution during stance and swing phase. One limb is in stance phase and the other limb is in swing phase during major portion of the gait cycle except during "Double support" which occurs at the end of both stance and swing phase.

in speed of walking the stance phase decreases, swing phase increases and the period of 'Double support' decreases (see Fig. 18.1).

- *Running*: It is defined as propulsion without period of 'Double support'. The ratio of stance phase and swing phase reverses and a 'Double swing' develops in running.
- *Center of gravity during gait*: The center of gravity (CG) is situated midway between the hips, a little in front of S2 vertebra during a normal gait. Energy expenditure is minimum if the center of gravity travels in a straight line. In a normal gait, the line of CG swings like a pendulum.

ABNORMAL GAITS

Due to Muscle Weakness, e.g. Poliomyelitis

Gluteus Maximus Lurch

Gluteus maximus muscle extends and laterally rotates the hip. To maintain the center of gravity, the pelvis remains backwards during the mid-stance phase because of this action.

If the gluteus maximus is paralyzed, this maintenance of hip in extension is not possible, instead the body swings backwards everytime when the weight is borne. Thus, it results in a gluteus maximus lurch.

Quadriceps Lurch

In quadriceps paralysis, there is loss of active extension of the knee. During the stance phase, which begins with heel strike and ends with heel off and toes off, the knee has to remain in extension. This is not possible in paralysis of quadriceps. So, a patient locks the knee in extension and bends forwards to support the front of the thigh with the hand. This results in hand knee gait or quadriceps lurch.

High Stepping Gait of Foot Drop

A normal gait begins with heel strike of stance phase. In foot drop, because of the paralysis of the dorsiflexors of the ankle and the foot, the heel strike is not possible and in order to clear the ground, the patient lifts the leg high and when the foot is brought down, the toes touch the ground first, before the heel. This results in a toe to heel gait, which is exactly the opposite of the normal heel to toe gait. This is known as a high stepping gait.

Calcaneus Gait

This occurs due to paralysis of the gastrosoleus. All the steps of stance phase except heel strike are affected. During the later part of stance, the weight is borne by the calcaneum and the foot rotates outwards in order to complete the stance phase. This results in a calcaneus gait.

Due to Changes in the Bones and Joints

Stiff Hip Gait

This type of gait is seen in ankylosis of the hip. The patient is not able to flex the hip during the swing phase. To complete the swing phase of gait cycle, he lifts the pelvis on that side and circumducts the involved leg. This results in a circumductory stiff hip gait.

Stiff Knee Gait

It is difficult to appreciate stiff knee gait because the changes that take place are subtle and need careful observation. The toe off phase is affected. To clear the ground for initiation of swing and for heel strike after completion of swing, a small degree of circumductory movement is required. This can be made out only by careful observation.

Short Leg Gait

When shortening of the limb is more than an inch, an appreciable limp develops. The shoulder on the opposite side dips down and lifts up because of upward and downward movement of the pelvis during weight-bearing.

Due to Abnormalities in the Nervous System

Scissors Gait

Due to the spasm of the adductors, the legs get crossed in front of each other during walking, resulting in classical scissoring. This is known as scissors gait and is seen in spastic paralysis of cerebral palsy.

Festinant Gait

Seen in Parkinsonism and is characterized by short strides. The feet barely clear the ground.

Stamping Gait

Seen in conditions which result in sensory loss, e.g. peripheral neuritis, tabes dorsalis, etc.

In this type of gait, because of sensory ataxia, patient stamps the feet to the ground.

Drunkard's Gait

Lesions of the cerebellum result in motor ataxia. Patient is not able to maintain motor balance and walks in an irregular path. This results in a Drunkard's gait.

Hemiplegic Gait

This is a classical gait seen in hemiplegia. The shoulder is adducted, the elbow and the wrist are flexed and the involved lower limb is circumducted during the swing phase of gait cycle.

Antalgic Gait

Any painful condition in the limb, which causes increase in pain during weight bearing, results in this type of gait. The condition may vary from a small thorn prick in the foot to tuberculosis of the hip. The stance phase is affected and on weight bearing, the patient immediately shifts the weight to the opposite normal limb because of pain. This results in a short and incomplete stance and an antalgic gait.

TRENDELENBURG GAIT (FIG. 18.2)

This is a type of gait that occurs when the abductor mechanism which is responsible for abduction at the hip is affected. The mechanism has three components:

a. The *fulcrum* which is the head of the femur
b. The *lever* which is the neck of the femur
c. The *power* which is the gluteus medius

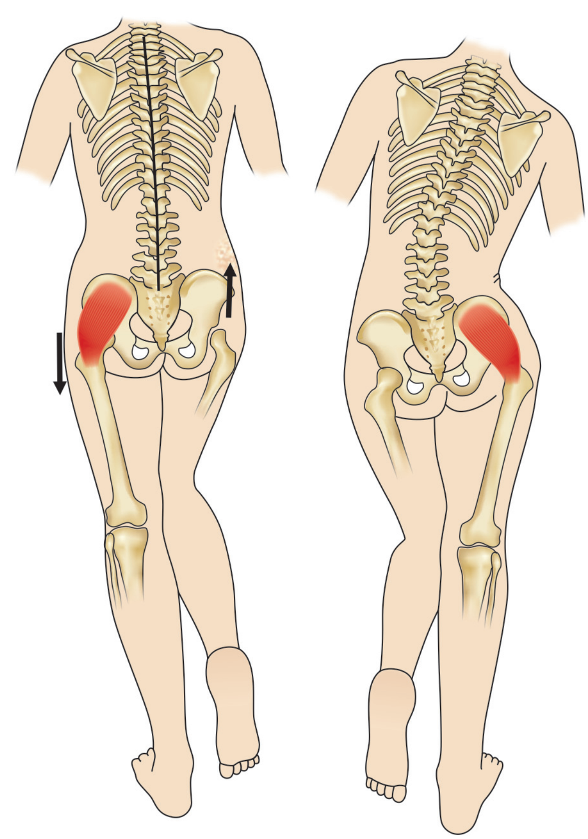


Figure 18.2
Changes occurring in the hip in 'Trendelenburg gait'. In this illustration the right hip is the involved hip and left hip is normal.

Any abnormality of these, occurring alone or in combination disturbs the function of abductor mechanism and results in this type of gait. The fulcrum is disturbed in dislocation of the hip. The lever is disturbed in fracture of neck of the femur. The power is disturbed in paralysis of gluteus medius.

Pathomechanics of Trendelenburg Gait

The abductor mechanism has a two-fold function. Acting from above, it abducts the limb and acting from below, it lifts the pelvis. On weight-bearing, the limb is fixed and it acts from below causing lifting of the pelvis. So, when the pelvis is lifted up, the anterior superior iliac spine on the opposite side moves up.

When the abductor mechanism is affected, this lifting of pelvis does not take place. Instead, the pelvis sags on the opposite side, when weight is borne on the affected limb. This causes the anterior superior iliac spine on the opposite side and the shoulder on the same side to dip down for maintaining the balance. A characteristic lurch develops on the affected side resulting in Tredelenburg gait.

When the abductor mechanism is affected bilaterally, a bilateral Trendelenburg gait or a Waddling gait results.

19 Amputations

- *Definition*
- *Indications*
- *Types of amputations*
- *An ideal stump of amputation*

"Amputation is one of the meanest yet one of the greatest operations in surgery. Meanest when resorted to while better things could have been done. Greatest when it is done to save the life of an individual."

DEFINITION

Removal of a limb or an appendage of the body surgically, is known as amputation.

INDICATIONS

The six d's for amputation are as follows.

i. A **d**ead limb, e.g. vascular gangrene.
ii. A **d**ying limb, e.g. TAO, frost bite, etc.
iii. A **d**estroyed limb, e.g. crush injury.
iv. A **d**enervated limb,e.g. hereditary sensory neuropathy, whole arm type of brachial plexus injury.
v. A **d**angerous limb, e.g. malignant bone tumor, gas gangrene.
vi. A **d**eformed limb.

In all the above situations, either the function of the limb is lost or the limb poses a threat to the life of an individual. Hence, there is a need for amputation.

TYPES OF AMPUTATIONS (FIG. 19.1)

General Amputations

- A ray amputation
- Below knee amputation
- Above knee amputation
- Below elbow amputation
- Above elbow amputation
- Disarticulation of hip, knee, etc.
- Hind-quarter and Fore-quarter amputation

Specific Amputations

- Gille's amputation
- Lisfranc's amputation
- Chopart's amputation
- Syme's amputation
- Pirogoff's amputation
- Kruckenberg's amputation

Gille's Amputation

It is a transmetatarsal amputation done in the foot. It preserves the function of weight bearing. Toes off phase of stance is lost. Needs special shoes with toe inset filled for a good cosmesis and to overcome the deficit of push off.

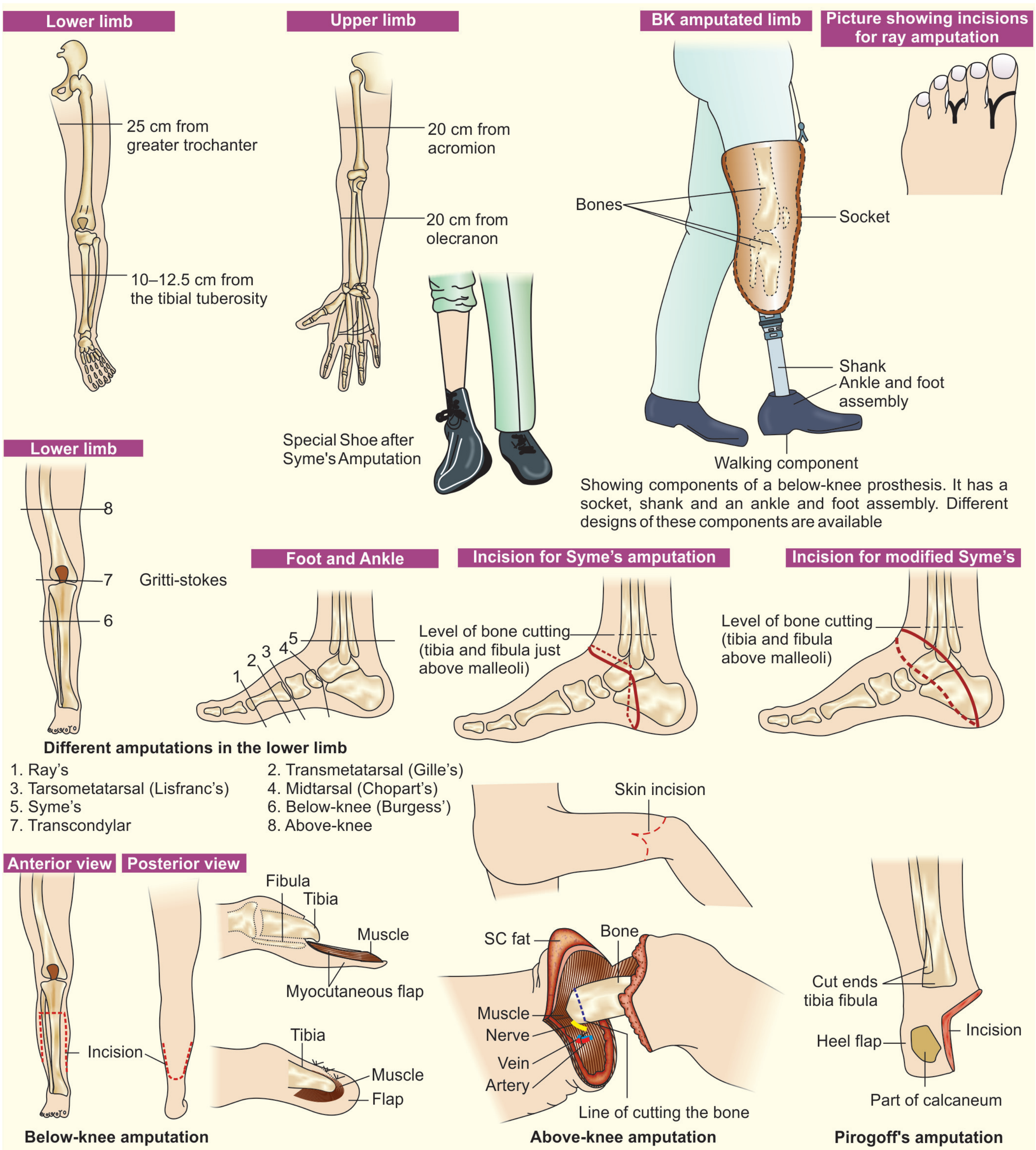


Figure 19.1

Different types of amputations, their levels, the ideal length of the stump and some prosthesis.

Lisfranc's and Chopart's Amputation (Ref: Foot injuries pages 61 and 63)

These amputations result in deficits in gait. Propulsion stops at foot flat stage of stance phase of gait resulting in deficits in mid-stance, heel off and toe off stages. ***In fact, Chopart's amputation can result in foot drop because of lack of muscle and tendon attachments.*** Weight bearing function is preserved. Special shoes with anterior fill, is used for cosmesis. Gait is affected.

Syme's Amputation

Named after James Syme , Professor of surgery in Edinburgh, who described this amputation in the year 1843, in London and Edinburgh Monthly Journal of Medical Science. It is an excellent amputation through the ankle, which retains the function of weight bearing because of intact heel pad. It results in a bulbous stump. To reduce the mediolateral diameter and the bulbous nature of the stump, trimming of the malleoli (Mazet 1968; Sarmiento 1972) and supramalleolar resection (Elmslie 1924) have been recommended.

A classical Syme's amputation is performed by:

a. Subperiosteal dissection and excision of the calcaneus.
b. Ligating the posterior tibial artery distally thereby preserving the blood supply to the heel pad.
c. Resection of the proximal tibia at the level of the dome. (Some do not recommend this step i.e. step c of classical Syme's)

A special prosthetic shoe is necessary after Syme's amputation. Weight bearing function is preserved. But, all the stages of stance phase of gait are affected.

Pirogoff's Amputation

In this modification, the calcaneum is resected partly and turned 90° upwards towards the tibia. This increases the length of the stump.

Kruckenberg's Amputation

This amputation was described by Kruckenberg and Putti. The amputation involves 'forcepisation' of the amputated stump of the forearm into radial and ulnar pincers (bifid forceps) resulting in two antebrachial fingers. These two pincers open and close in pronation and supination movements of the forearm. The length of the pincers can be varied from 7–12 cm. Longer the pincer length, greater is the interdigital defect which may necessitate a skin coverage procedure. Also, the strength of the grip decreases. The amputation is unsightly, but highly efficacious functionally with retained sensation. Can be fitted with a cosmetic hand prosthesis.

AN IDEAL STUMP OF AMPUTATION

It should fulfill the following criteria

1. Long enough to fit a prosthesis
2. Good power
3. Good sensation
4. Good blood supply
5. Good soft tissue cover
6. No neuroma
7. No bad scarring
8. No infection
9. Conical shape
10. Proximal joint should be normal

Recommended Ideal Length of the Stump

1. In below-knee amputations, 10.0–12.5 cm from the Tibial tuberosity.
2. In above-knee amputations, 22.5–25.0 cm from the greater trochanter.
3. In above and below elbow amputations, 20.0 cm from the Acromion process and the Olecrenon process respectively.

Note: *These stump lengths recommended, are not constant. The length varies depending on the length of the limb. Basically, it gives a rough idea as to how much length of the stump is desirable for fitting a prosthesis.*

General principles to be observed during the procedure of amputation:

1. Tourniquet is always used except in a case of vascular disease.
2. Flaps are marked properly before the skin incision is made.
3. Designing equal antero-posterior flaps is desirable.
4. The procedure should aim at designing an ideal stump.
5. Skin division is the farthest, followed by fascia, muscle and the bone. Each of them is divided, a

little more proximal, to the preceding tissue in the above mentioned order.

6. Proximal part of the flap should contain full thickness of the tissue, from skin to bone whereas the distal part of the flap should contain only skin, subcutaneous tissue and the fascia to allow good closure of the wound.
7. Periosteal stripping should not exceed the required extent as this will result in formation of sequestrum at the end of the stump (crown sequestrum).
8. Sharp spikes of bone are trimmed and made round.
9. The nerves are pulled a little and then divided in order to allow retraction.
10. The major vessels are to be perfectly ligated.
11. Tourniquet is always released before closure and hemostasis achieved.
12. Wound always closed over a drain.
13. Good stump bandage for support to be given (immobilizing the joint above, e.g. the knee and the elbow in BK and BE amputations respectively).
14. Regular stump exercises are to be done to prevent joint contractures.

20 Arthroscopy and Total Joint Arthroplasty

- *Arthroscopy*
- *Total joint replacement*

ARTHROSCOPY

Arthroscope is an instrument which allows viewing of the interior of a joint. A video camera attached to the eyepiece of the scope is connected to a monitor and the picture is seen on a screen. When used only for viewing, the procedure is known as diagnostic arthroscopy. When surgical procedures is performed under the guidance of the scope, the procedure is called arthroscopic surgery.

Evolution of Arthroscopes

It was Professor Kenji Takagi who first viewed the interior of a cadaveric knee, in the year 1918, using a cystoscope and later developed a scope known as Takagi scope in the year 1931. Dr Philip Kreuscher recommended the use of arthroscope for recognition and treatment of meniscal lesions in the year 1925. Credit goes to Dr Masaki Watanabe for not only developing the first modern arthroscope in the year 1958, but also performing the first ever arthroscopic surgery of excising a xanthomatous giant cell tumor as well as performing, meniscectomy, in the year 1962. In mid 80's, the videoscope and motorized arthroscopic instruments were developed and advanced techniques started evolving. These days, many surgeries are being performed using arthroscope. Arthroscopes of different sizes and designs are available for all the joints. Techniques are also highly advanced.

Components of an Arthroscope

The basic component of an Arthroscope is an endoscope with a metal casing. The scope has various degrees of viewing angles of 0°, 30°, 70°, etc. at the distal end and an eyepiece (to which a video camera can be connected) at the proximal end. It also has a portal for the attachment of a light source. The inside of a scope consists of a series of lenses and optical fibers which transmit light from the light source to the interior of the joint (Fig. 20.1).

Technique

The procedure is carried out under general/regional anesthesia with full muscle relaxation. Muscle relaxation is necessary as it allows good maneuverability of joints during the procedure. For proper visualization, the joint is distended with infusion of fluid. For the purpose of diagnosis, a single portal is generally sufficient. But, for arthroscopic surgery additional portals for insertion of specialized instruments are necessary. Insertion portals for the scope as well as for the instruments are planned depending on the nature of the lesion (Fig. 20.2). The joint is viewed in a systematic manner. At the end of the procedure, the joint is thoroughly irrigated, the skin wounds are sutured and a pressure bandage is given.

Figure 20.1
Arthroscope along with some accessory surgical instruments.

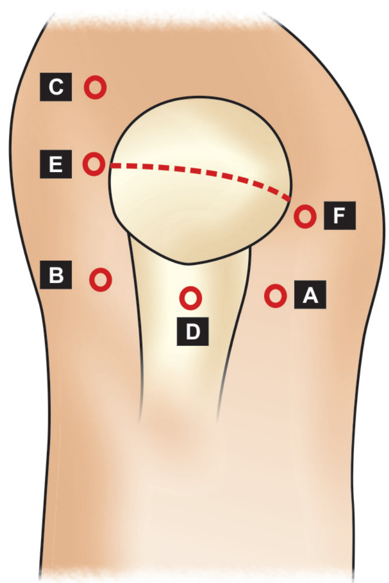


Figure 20.2
Different portals of entry for knee arthroscopy. A, B, C are standard portals; D, E, F are additional portals. All are marked. (A) Anteromedial (B) Anterolateral (C) Superolateral (D) Transpatellar (E) Mid patellar lateral (F) Mid patellar medial.

Advantages of Arthroscopic Procedures

1. Less invasive, less morbid and less painful.
2. Day care procedure with no hospital stay.
3. Early mobilization possible.
4. Direct visualization of the abnormality is possible.
5. Dynamic picture of the joint is recordable.
6. Good cosmesis.

Limitations of the Arthroscopic Procedures

Not indicated in advanced involvement of a joint in case of osteoarthritis, infective conditions, major joint injuries, etc.

Complications

1. Infection
2. Hemarthrosis
3. Reflex sympathetic dystrophy
4. Thrombophlebitis

Common Arthroscopic Procedures

Synovium
1. Synovial biopsy
2. Synovectomy

Articular cartilage
1. Excision of loose bodies, e.g. osteochondritis desicans.
2. Shaving of the degenerated cartilage, e.g. osteoarthritis.

Bone
1. Correction of patellar tracking
2. Biopsy of an intra-articular lesion

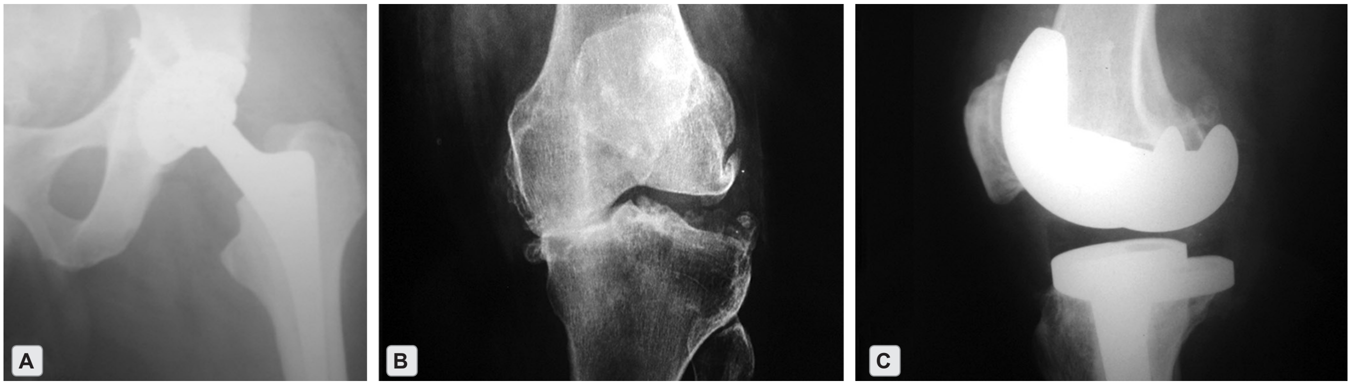
Ligaments
1. Primary repair, e.g. MCL.
2. Secondary reconstruction, e.g. ACL.

Meniscus
1. Partial or complete meniscectomy
2. Meniscal repair

Lavage
Thorough irrigation and drainage, e.g. infective arthritis, osteoarthritis

TOTAL JOINT REPLACEMENT (ARTHROPLASTY)

When both the articular surfaces of a joint are replaced by an artificial joint (Prosthesis), the procedure is known as total joint arthroplasty (Figs 20.3A to C). A total joint arthroplasty converts a destroyed, disorganized, painful joint which has lost function into a near normal, well organized, painless joint with good function. This is



Figures 20.3A to C

(A) Uncemented total hip replacement; (B and C) Total knee replacement for osteoarthrosis.

achieved by clearing the damaged portion of the bone and inserting an appropriate artificial prosthesis. Adequate preoperative planning is necessary for this surgery.

Evolution

Sir John Charnley is considered as the pioneer of total joint replacement, especially the hip. He popularized the concept of low friction arthroplasty in 1960's by designing a metal femoral stem component and a polyethylene cup component for the acetabulum. He did this work in the Center for hip surgery, Wrightington; England. He used polymethylmethacrylate, the bone cement which he initially borrowed from his dental friends. Cement acted as a filler and helped in the firm fixation of the components. The procedure proved to be very successful and subsequently, many proshetic designs were developed. Because of the complications faced at the time of insertion of the cement and problems faced at the time of removal of a failed prosthesis, uncemented prosthetic designs developed. These designs were later modified into hydroxyapetite coated prosthesis aiming at firm fixation at bone metal interface. Currently, the use of cemented prosthesis is restricted to elderly people and uncemented prosthesis is always preferred in young individuals.

The year 1968 saw the development of a successful knee prosthesis. A Canadian Orthopedician Frank Gunston from John Charnley's center, replaced the knee with a metal and polyethylene component which were fixed to the bone by bone cement. In 1972, John Insall developed a design of total knee which became the prototype of all the total knees available these days.

Total ankle, total shoulder and total elbow are the other prostheses that are available these days, for respective joint replacements.

Success of joint replacement depends on preoperative planning and accurate intraoperative execution of the preoperative plan. The alignment of both the components of a total joint prosthesis should be perfect.

Computer assisted joint replacement surgery is also practiced in some centers for more accuracy.

Complications

Some of the complications encountered during joint replacement are as follows:

1. Infection
2. Hemorrhage
3. Deep vein thrombosis (DVT) and pulmonary embolism
4. Loosening of the implant
5. Dislocation
6. Nerve palsies

Postoperative Rehabilitation after Joint Replacement Surgery

1. The patient is mobilized on the bed (joint movements) as early as possible after the initial pain subsides.
2. Weight bearing in hip replacement surgeries is usually delayed when compared to knee replacement surgeries and is related to the type of prosthesis used (cemented and uncemented).
3. A walking aid is always given during the initial period of weight bearing.
4. The patient is advised to avoid strenuous activities so as to increase the longevity of the prosthesis.

21 Implants and Instruments

- *Surface implants*
- *Intramedullary implants*
- *Biodegradable implants*
- *General instruments used in orthopedics*
- *William Arbuthnot Lane*
- *William Ferguson*
- *Designs of limb lengthening apparatus*

INTRODUCTION

Instruments are devices which are used to perform surgical procedures. Implants are the devices which are placed (planted) in the human body. When implants that are used to replace a diseased/damaged part totally or partially, are termed as prostheses (singular-prosthesis).

Prostheses are designed for permanent use. They are removed only when they fail.

Whereas, implants (other than prostheses) serve only as an internal splint for a specified period within which the fracture healing occurs, following which they have no role. They may have to be removed. The implants once removed should not be reused.

Materials Used for Orthopedic Implants

Alloys of stainless steel 316 L and titanium as well as more recently biodegradable materials like polyglycolic acid, polylactic acid and polydioxanone are used for manufacturing orthopedic implants.

Desirable Properties of an Implant

1. The modulus of elasticity of the implant should be close to that of the bone. Biodegradable implants are the closest. Among the alloys, Titanium is the closest (Fig. 21.1).
2. Should be biocompatible.
3. Should be chemically stable.
4. Should have good ductility.
5. High fatigue resistance.
6. High mechanical strength (lacking in biodegradable materials).
7. Nontoxic.
8. Noncarcinogenic.

Types of Implants

Basically, there are two types:

a. Surface implants, e.g. plates and screws.
b. Intramedullary implants, e.g. nails.

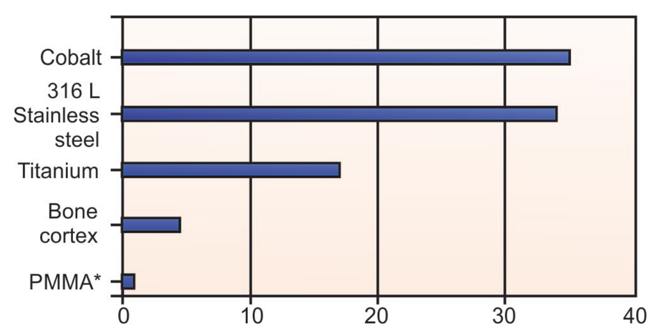


Figure 21.1

Modulus of elasticity of different materials. Titanium is the closest when compared to other materials. *PMMA—Polymethyl methacrylate.

Types of Prosthesis

Two Types

a. Those which replace components of bone, e.g. prostheses used for joint replacement (Fig. 21.19).
b. Those which are used to replace whole structures, e.g. intervertebral disk, scaphoid, lunate, etc.

Implant Selection

Healing is a natural process that is dependant on several factors. An implant design should not disturb this natural biological process of healing. Immobility at the site is one of the factors essential for healing and implants take care of this factor by providing immobility at the site.

None of the implants allow full weight bearing or full activity immediately after surgery. There is a postoperative protocol to follow. Failure to observe this, results in implant failure.

Following factors are to be taken into consideration while selecting an implant.

a. Age of the patient
b. Weight of the patient
c. Mental status and intelligence
d. Bone involved
e. Type of fracture
f. Quality of the bone
g. Associated injuries and comorbid factors
h. Foreign body sensitivity

In pediatric age group, unlike adults, implants are used only with definite indications. Less traumatic and easily removable implants are preferred, e.g. 'K'-wires, titanium elastic nails, etc.

Stronger implants are preferred in heavily built individuals.

Demand of the situation dictates the type of the implant to be selected (Table 21.1). (1) A transverse fracture is stable against axial compression but unstable against torsion and bending stress. (2) An oblique fracture is stable against torsion stress but unstable against axial compression and bending stress. (3) A spiral fracture is unstable against both axial compression and torsion stress. (4) A comminuted fracture is highly unstable against all the types of stresses. It entirely depends on the strength of the implant to maintain stability. (5) An open fracture in addition to the nature of bony injury, poses a challenge because of associated injury to soft tissue envelope.

When an individual is sensitive to stainless steel, titanium implants are preferred. These are also used when MRI is indicated during the postoperative and follow-up period, e.g. in spinal stabilization, surgeries around the hip when AVN is expected, etc. If stainless steel implants are selected, they do not allow MRI during follow up period because stainless steel interferes with the magnetic field generated during MRI.

Senility, alcoholism, mental illness may lead to implant failure because of failure to adhere to postoperative protocol. Associated comorbid factors also can contribute to implant failure.

Note: *It should always be remembered that the implants that are used to stabilize, have a great role to play, to maintain the correction achieved by surgery. They have no role to play, once the healing occurs***.**

Table 21.1

Implant selection.

In Lower Limb		
Type of fracture	*Instability against stress*	*Order of implant preference*
Transverse	Unstable against torsion and bending Stable against axial compression	1. Intramedullary interlocking nail 2. Plate fixation
Oblique	Unstable against axial compression and bending Relatively stable against torsion (when compared to transverse and spiral)	1. Intramedullary interlocking nail 2. Plate fixation
Spiral	Unstable against axial compression and torsion. Relatively stable against bending (when compared with transverse and oblique)	1. Plate fixation 2. Intramedullary interlocking nail
Comminuted	Unstable against all stresses	Either intramedullary interlocking nail OR plate fixation

Note: 1. This chart gives an indication in lower limb fractures only with respect to the nature of instability seen in different anatomical types of fractures. Other factors (refer text) such as age of the patient, site of the fracture, the bone involved, quality of bone, etc. also play a role in implant selection.
2. Both, intramedullary interlocking nailing as well as plate fixation, give stability against all the deforming stresses and can be employed for fracture fixation using closed OR open techniques. Load bearing nature of the plate and load sharing nature of the nail has to be kept in mind during selection.
3. In the upper limb, the fractures are subjected commonly to stresses of bending and torsion and less commonly to axial compression. Hence, plating is prefered to nailing. Again, other factors (refer text) are also considered in implant selection.

Implant Insertion

Insertion of an implant is an art. Correct handling of the implant is an absolute must during insertion. This skill has to be acquired from the very beginning itself. Meticulous attention has to be given to the use of proper instruments and the techniques of insertion. Techniques vary depending on the design of the implant and hence, specialized instruments exist. Improper handling increases the failure rate and complications.

Asepsis in the Operating Room

This is of utmost importance. There goes a popular saying which says that ***"You can spit into the abdomen but you cannot breathe over the bone"***. Meticulous measures for asepsis are to be observed in the orthopedic operation theater.

Infection following an insertion of an implant is a disaster. The implant gets rejected and the whole procedure, may end up in a failure. A center where the infection rate is less than 1% is ideal for orthopedic surgery. 1–3% is acceptable. But, above 3% is to be considered as not suitable for orthopedic surgery. A separate dedicated theater for orthopedic surgery is desirable.

SURFACE IMPLANTS

These are placed on the surface of the bone and hence, bear the load of axial stress (Fig. 21.13).

Screws

Screw is a device which converts a rotational force into linear motion (Fig. 21.2). The design of the screw enables it to bring about this movement when it is turned. Screws are used to fix surface implant to the bone. Specially designed screws are also used to fix small bone fractures, e.g. Scaphoid and Malleolus. Multiple screws are used to fix larger fragments, e.g. Fracture neck of femur.

The Screw Design (Figs 21.3 to 21.7)

i. *Head of the screw:* It is that part of the screw which has slot or socket for fixing an attachment to the device that applies a rotational force (screw driver). When the insertion is complete it gives resistance and prevents further motion.
ii. *Shank with threads:* This helps to gain purchase in the bone while gliding (while inserting) and helps in the firm fixation of the screw.
iii. The tip directs the screw.

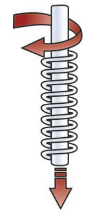
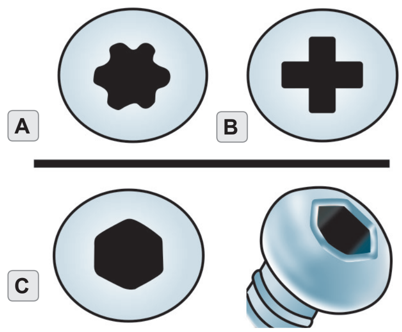


Figure 21.2
Rotational force being converted into a linear movement.



Figures 21.3A to C
Different types of screw heads: (A) Torx head; (B) Philips head, and (C) Hexagonal head.

The distance between the threads is called the pitch of the screw and the distance which the screw moves during one complete turn, is the lead of the screw. The pitch and the lead are equal, i.e. if the pitch is 1 mm the lead also is 1 mm.

The variation in the pitch of a cortical and cancellous screw is for the purpose of gaining good purchase in the cortical and cancellous bones, respectively. The cortical bone is a compact bone and the cancellous bone is a spongy bone. Hence, the difference in the pitch designs (Figs 21.5 and 21.6).

The screws are available in different lengths and different diameters.

To summarize, the different types of screws can be identified by looking at the thread design.

1. The differentiating point for a cortical screw from other types of screws is that it has threads throughout the shank (Fig. 21.4) whereas other screws do not.
2. The differentiating point for a cancellous screw is that it has threads only distally and the smooth shank without threads extends proximally up to the head of the screw (Fig. 21.5).
3. A cannulated cancellous screw, is similar to cancellous screw but has a central canal for insertion over a guidewire.

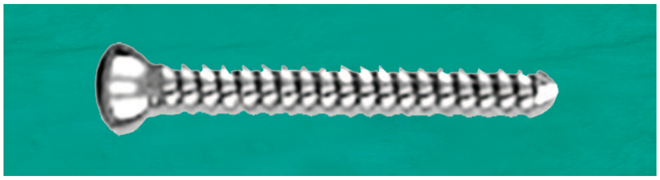


Figure 21.4

A cortical screw. It has threads along the length of its shank and has a smaller pitch.

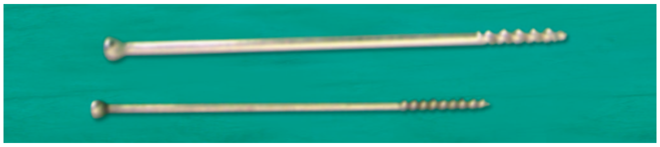


Figure 21.5

Cancellous screws. The bigger one is a 6.5 mm and the smaller one is 4.0 mm. They have threads only at the tip and a larger pitch. They are available in 16 mm and 32 mm thread lengths as well as in a fully threaded design.

Figure 21.6

A malleolar screw. This screw is used for fixation of the malleolus and has a conical sharp cutting tip, with threads of a different pitch as well as width, from that of a cortical and a cancellous screw. The extent of thread on the shank is more than that of cancellous screw, extending almost up to 1/2 the length of the shank.

Figures 21.7A to C

(A) Herbert screw (magnified). It is used in the fixation of the fracture scaphoid. This is a headless screw having threads of different pitch at the proximal and the distal ends. (B and C) Locking screw of a Locking Compression plate and interlocking nail respectively. Note the threads on the head for engaging and locking in the hole of the plate.

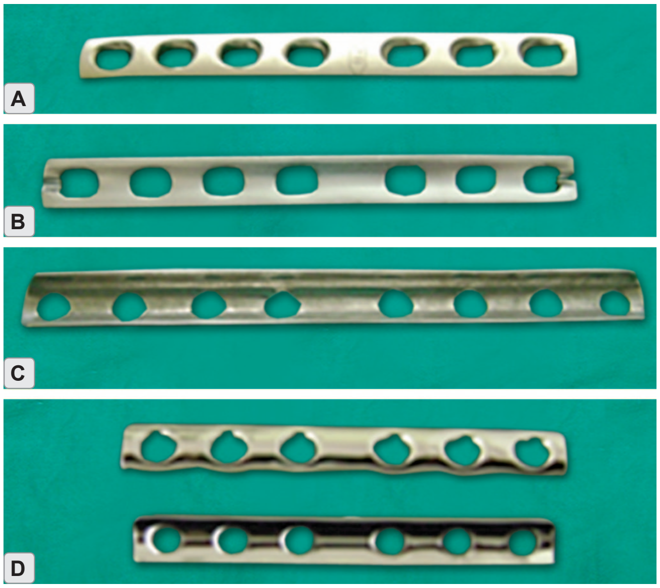
4. A malleolar screw looks similar to cancellous screw but it has a triangular cutting tip and threads extending more proximally, almost half the length of the shank (Fig. 21.6), when compared to a cancellous screw.
5. A scaphoid screw is a headless screw, with threads of different pitch at both ends (Fig. 21.7A).
6. A locking screw of an LCP has a head with threads on it (Fig. 21.7B).
7. A locking screw of an interlocking nail has threads throughout its length but these threads are of a wider pitch and lesser width when compared to a cortical screw (Fig. 21.7C).

Plates (Figs 21.8 to 21.11)

Plate is a surface implant with holes. It is placed on the surface of a bone and is fixed to the bone by screws. The outer surface is convex and the inner surface is concave to accommodate the cylindrical structure of the bone. Different designs of plates are available.

Dynamic Compression Plate (DCP)

The curvature of this plate is equal to 1/8 of a circle. The holes are distributed equidistant from a solid non-



Figures 21.8A to D

Dynamic compression plate, the semitubular plate and a 1/3 tubular plate. (A and B) DCP. Note the placement of holes with sloping edge. The curvature amounts to 1/8 the circumference of a circle. (C) Semitubular plate. The curvature amounts to 1/2 the circumference of a circle. (D) 1/3 Tubular plate. The curvature of a 1/3 tubular plate amounts to 1/3 the circumference of a circle.

fenestrated center of the plate. The holes are oval in shape and the distal edges of the oval hole have a slope towards the center, in order to bring about compression effect, when the screws are tightened.

The DCP was very popular in 1980's. Now, it is not used as a dynamic compression plate. When used, it is applied as an ordinary plate and not as a Dynamic Compression Plate.

It was thought that fractures unite early, because of compression force generated due to the inherent design of the plate as well as the method of insertion of the screws. After fixation, patients were mobilized early and fracture disease was prevented. Now, it is not used because the principle of compression fixation of fractures using DCP, is known to cause secondary changes which are detrimental to the bone. Also, it is a well established fact that union without callus is far inferior to union with callus and remodeling, with respect to strength of the bone.

Some of the complications/changes which proved detrimental to the use of DCP were as follows:

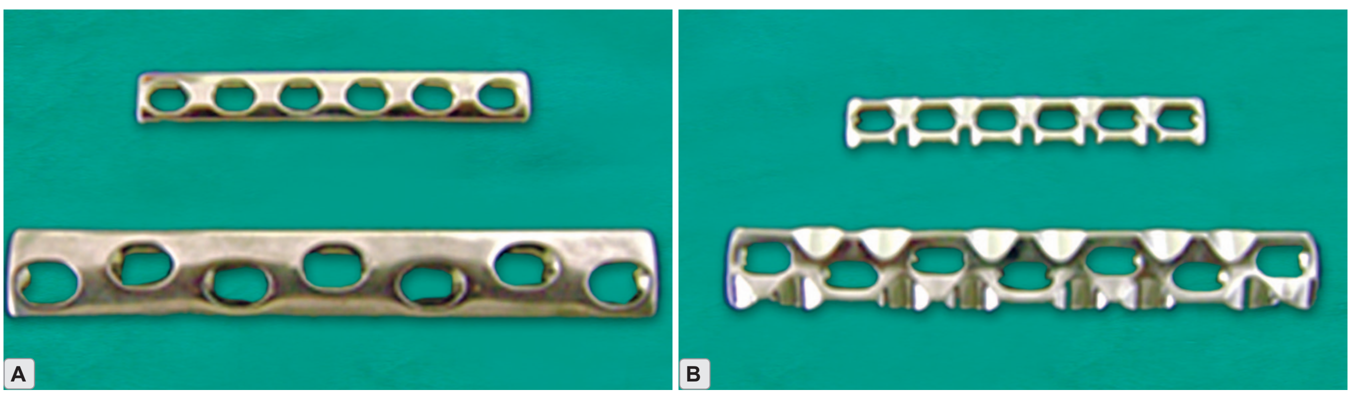
i. Union without callus formation in the form of direct substitution by the creeping trabeculae, caused refracture after removal of the plate.
ii. Stress fractures occurred at the ends of the plate.
iii. During removal of the plate, the bone under the plate was found to be avascular.

1/3 Tubular and Semitubular Plates

These are used mainly to fix the fractures in the supportive bones like ulna and fibula.

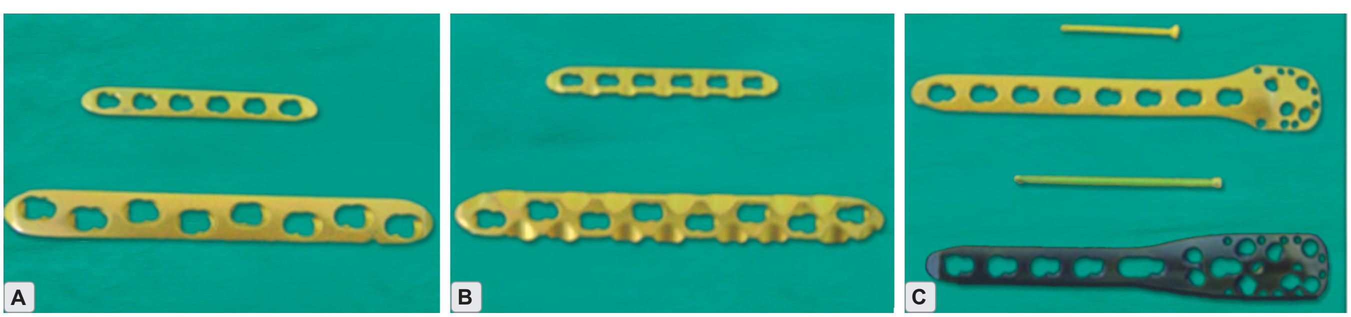
Limited Contact, Dynamic Compression Plate: LC, DCP (Figs 21.9A and B)

To overcome the disadvantages of DCP, the design was changed. Grooves were designed on the concave under-surface of the plate for blood vessels to grow. The holes were distributed evenly all along the plate



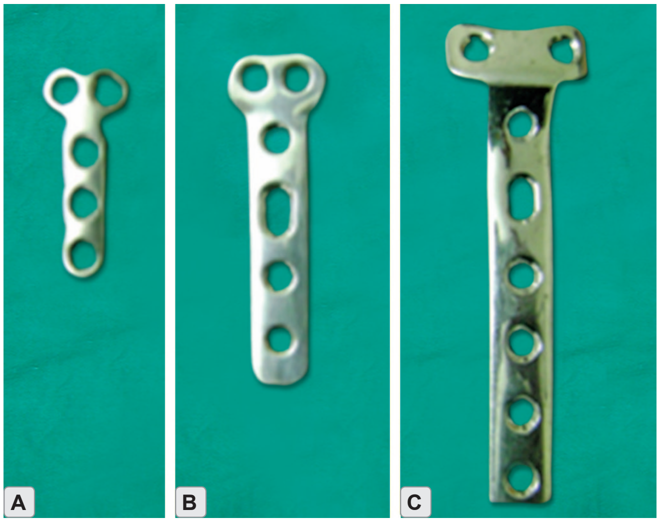
Figures 21.9A and B

LC, DCP. Note the changed design with grooves on the under surface of the plate and uniform distribution of holes for insertion of screws. Also note, that the changed design of the under surface of the plate, allows limited contact with the bone. This limited contact allows vascular ingrowth and prevents avascular changes occurring underneath the plate.



Figures 21.10A to C

(A and B) the different sizes of LCP. Note the options for locking as well as nonlocking for the head of the screw; (C) Proximal humerus LCP with a variety of options for the insertion of screw with respect to size and type and direction of the screw. Note that the screw in green color, has a head with threads for locking.



Figures 21.11A to C

'T' type Buttress plates for different injuries. (A) Thumb metacarpal (Rolando's fracture); (B) Lower end of radius (Smith's fracture); (C) Tibial condyles (Fender fracture).

to prevent concentration of stress, without taking into consideration a central reference point. Currently, these plates are being used, in place of DCP.

Locking Compression Plate (LCP) (Figs 21.10A to C)

These are specially designed to fix fractures in osteoporotic skeleton. The problem faced in an osteoporotic skeleton is that of not having a firm purchase in the bone. Without firm purchase, the screws will become loose and failure of fixation occurs. The design of the holes in LCP gives an option to exercise locking or nonlocking. At the same time, it gives an option for placing the screws at right angle to the surface of the bone or at an oblique angle. These options, improve the firmness of fixation. In practice, this plate serves as an internal fixator.

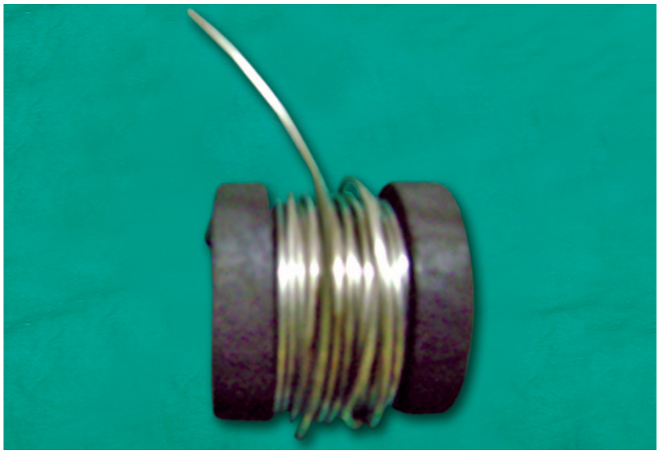


Figure 21.12

Stainless steel—SS wire used for tension band and circlage wiring in fixation of fractures. Available in different gauges.

SS wires

These are stainless steel wires, used in the fixation of comminuted fractures, in tension band wiring, circlage wiring, etc. (Fig. 21.12).

INTRAMEDULLARY IMPLANTS

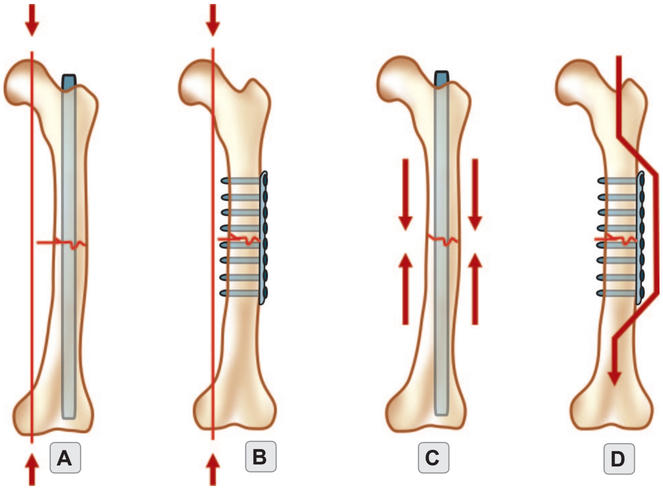
These implants are placed in the medullary canal of a bone and hence share the stress of axial loading (Figs 21.13A to D).

These are basically classified as rigid nails, hollow nails and elastic nails (Figs 21.14 and 21.15).

Rigid Nails

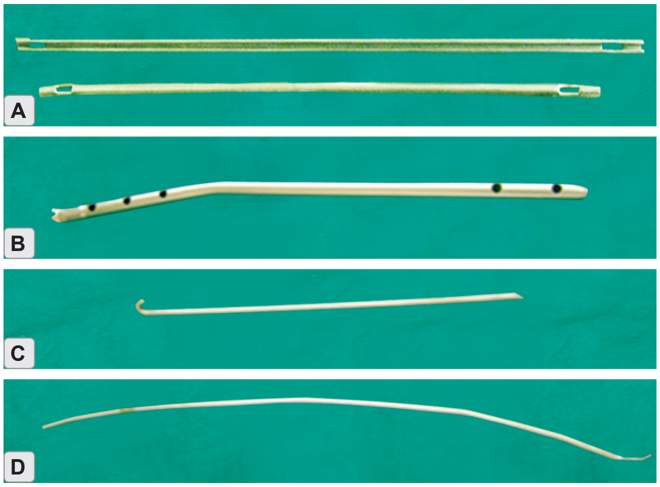
These nails are solid rods and do not have a canal. They have a good tolerance against the stress of axial loading but poor tolerance against bending stress. Thus they easily break when subjected to bending stress, e.g. unreamed interlocking nails.

They are indicated in open fractures and badly comminuted fractures.



Figures 21.13A to D

The bending moment arm in a nail versus a plate fixation. The moment arm of a plate is longer when compared to a nail. Hence, the plate is subjected to greater stress when compared to a nail. Thus, the plate acts more as a load bearing device and the nail acts more as a load sharing device.



Figures 21.14A to D

(A) Kuntscher nail; (B) Tibial interlocking nail; (C) Rush nail; (D) Ender nail.

Hollow Nails

These nails have a central canal/slot. They are not as strong as rigid nails. But, they have a good tolerance for bending stress and do not break easily, when subjected to such a stress.

These are indicated in all closed noncomminuted fractures.

Elastic Nails

These are thin solid rods and on their own, do not have any strength to resist axial stress, but show excellent adaptiveness for bending stress. Multiple nails have to be used to get sufficiently rigid fixation and to resist the stress of axial loading, e.g. ender nails, titanium elastic nails, etc. Indicated in pediatric long bone fractures and in elderly, osteoporotic, long bone fractures.

Kuntscher Nail or K-nail

This is an intramedullary nail designed by Gerhard Kuntscher, a German surgeon, during II world war. It is a hollow slotted nail, which is clover leaf shaped in cross-section. It has extraction slots at either ends. It fixes the fracture shaft femur based on the principle of three point fixation (Fig. 21.15). The three points being the cancellous portion of the intertrochanteric bone, the isthmus and the cancellous portion of the intercondylar bone. It was in use till mid 80's for almost 40 years until interlocking nails became popular. Even now, these nails are being selectively used.

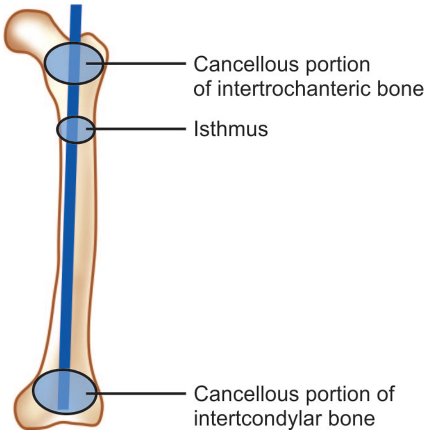
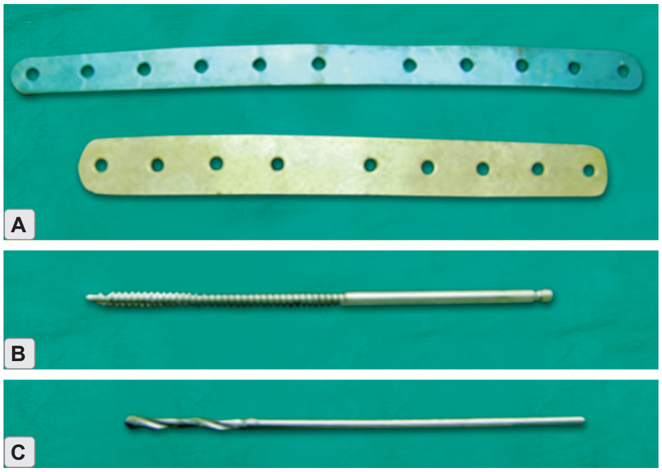


Figure 21.15

Three point fixation of the Kuntscher intramedullary nail.



Figures 21.16A to C

Some of the instruments used for plating: (A) Bending template to assess the curvature of the bone and bend the plate accordingly; (B) Tap for cutting threads after drilling hole in the bone; (C) Drill bit to drill a hole.

Gerhard Kuntscher, knew that the nail did not give much rotational stability and came out with Detensor nails with perforated ends meant for locking bolts. This, became the precursor of all the interlocking nails of today.

Rush Nail

This is a rigid nail designed by Lesley Rush and Lowry Rush, American orthopedic surgeons in 1930's. These are rigid nails having a beveled tip and a curved hook like head with a bevel on it.

The direction of bevel on the head corresponds to the direction of the bevel at the tip, so that the surgeon knows the direction of the bevel at the time of insertion of the nail, after its entry into the madullary canal of the bone.

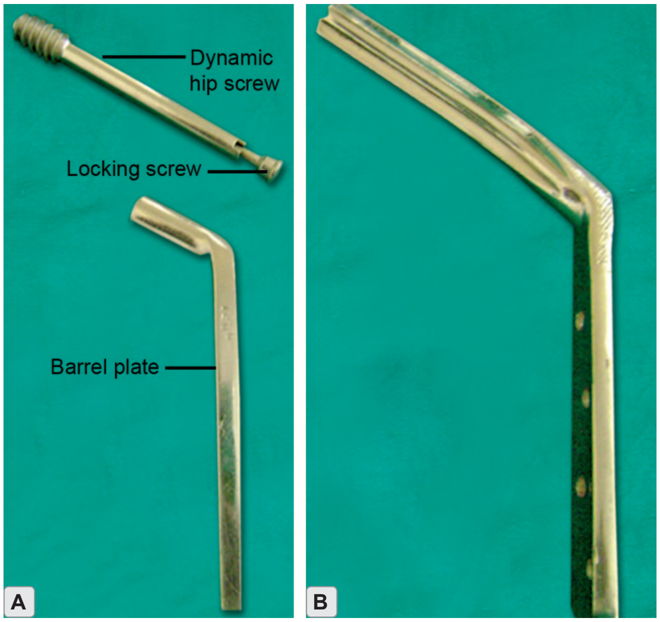
They were used to fix the fracture of the forearm bones and were supposed to work on the principle of trifocal buttressing, i.e. according to Rush 'A vase of flower'.

BIODEGRADABLE IMPLANTS

These implants have a modulus of elasticity very close to that of the bone, even better than the titanium. But, they exhibit a very poor strength to axial and bending stress. Hence, currently they are used only in fractures involving small bones/small fragments, e.g. malleolar fracture, metacarpal fracture, lateral humeral condyle fracture, osteochondral fracture, etc. The greatest advantage is that they get degraded and absorbed in 6 months to 1 year. Disadvantage is the severe tissuse reaction with signs of inflammation at the time of biodegradation process, mimicking acute infection.

Dynamic Hip Screw and a Barrel Plate (Fig. 21.17A)

This is an implant, which is used to fix the intertrochanteric and some types of subtrochanteric fractures of the femur. The implant consists of a cannulated hip screw, a barrel plate and a locking screw. It works on the principle of rigid internal fixation. Firm fixation is brought about when the locking screw, which locks the hip screw and the barrel together, and is tightened. Compression is also brought about when the patient starts bearing weight and the hip screw begins to slide within the barrel plate.



Figures 21.17A and B

(A) Dynamic hip screw and barrel plate; (B) Jewett nail.

Jewett Nail (Fig. 21.17B)

This is a fixed angle nail plate device used to fix the intertrochanteric fracture. It has a cannulated triflanged nail fixed to a plate, at a specified angle. These are available with varying angles from 120°–145° and chosen according to the angle desirable at fixation. Now used very selectively.

Cannulated Cancellous Hip Screw

Fixation of fracture neck of the femur started with the use of Smith Peterson Nail. To begin with, this was a solid implant with 4 flanges. Later, this was modified into a triflanged nail. Johanssen modified the nail by introducing a central canal into the nail. In current practice, the cannulated cancellous screw is the standard implant that is used to fix the fracture neck femur. A minimum of two and a maximum of three screws can be used (Fig. 21.18).

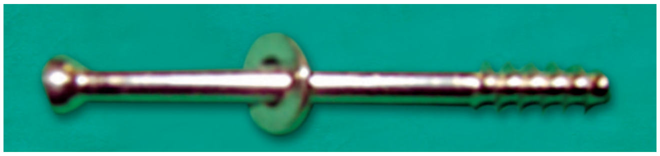


Figure 21.18

Cannulated cancellous hip screw with a washer, for the fixation of fracture neck of the femur.

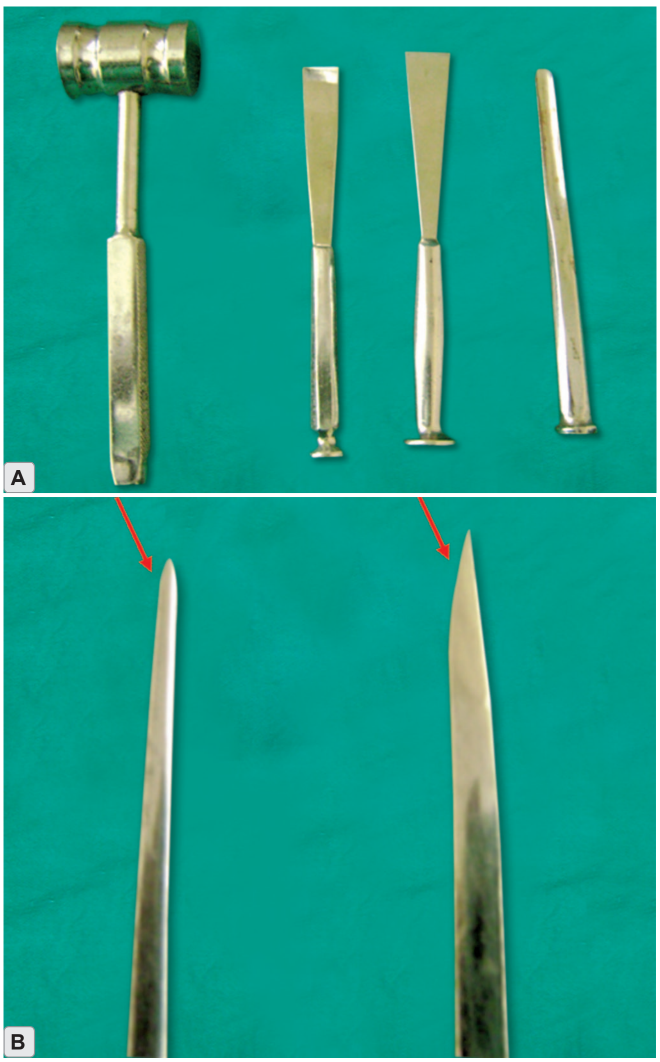
Figures 21.19A and B

(A) An Austin Moore's prosthesis (B) a Thompson's prosthesis. These are commonly used in fracture neck of femur for replacement hemiarthroplasty. Note the fenestrations in the stem of Austin Moore's prosthesis for the ingrowth of bone and initiation of a self-locking process. This is used when calcar femorale is sufficient. When calcar femorale is deficient, Thompson's prosthesis with bone cement is used.

GENERAL INSTRUMENTS USED IN ORTHOPEDICS (FIGS 21.20 TO 21.29)

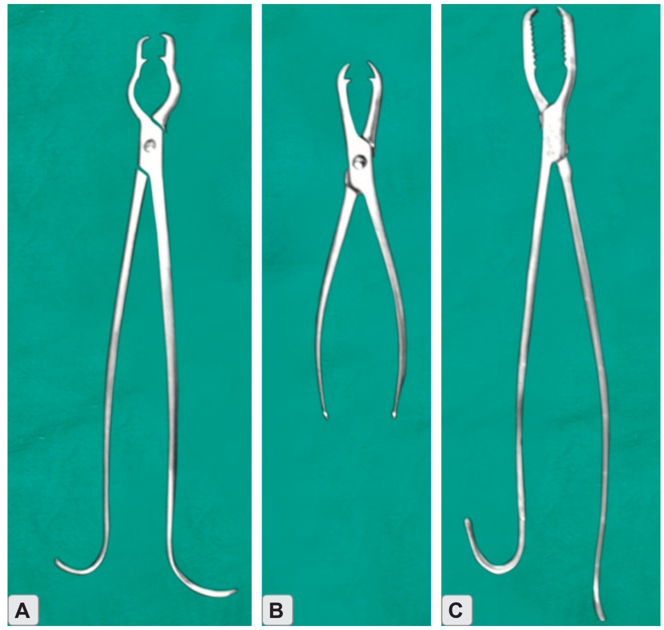
Mallet

A mallet is used to deliver a blow either to drive an implant or to drive an instrument, e.g. in the insertion of a nail or a prosthesis with the help of an impactor (Fig. 21.25); in osteotomies to drive an osteotome; in bone grafting to drive a chisel, etc.



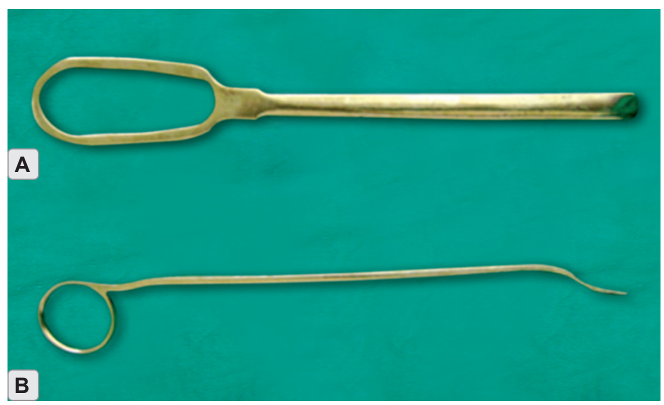
Figures 21.20A and B

(A) A mallet, a chisel, an osteotome and a bone gouge from left to right respectively. (B) The difference between the cutting edge of an osteotome and a chisel. An osteotome has beveling on both sides (left) where as a chisel has a beveling on one side (right).



Figures 21.21A to C

The three types of bone holding forceps. (A) Lane's bone holding forceps. (B) Ferguson's lion toothed (or Jawed) bone holding forceps. (C) Hey Groves bone holding forceps.



Figures 21.22A and B

Two types of bone levers. These are used to lever the bone free from its soft tissue. (A) Bristow's type. Sir Walter Rowley Bristow devised this lever. (B) Trethowen's type. Sir William H Trethowen devised this lever. Hence the names.

Figure 21.23

A periosteal elevator used to strip the periosteum from the bone. Note the serrated portion in the handle which is used for placing the thumb, in order to have a firm grip.

Figure 21.24

A bone hook which is used to hook the bone and draw it up from within the soft tissue.

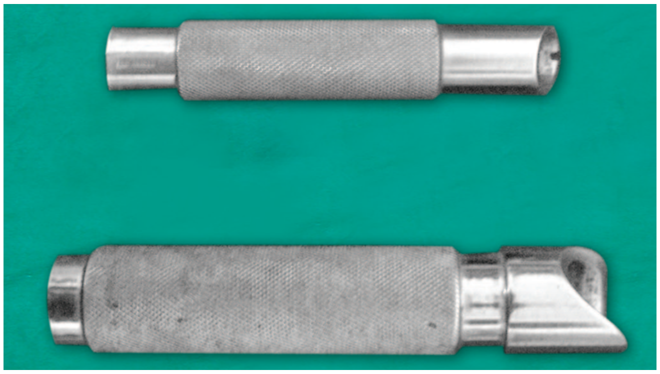


Figure 21.25

Two types of nail impactors. Note the slotted and the hollow design at one end for the nail and a knob at the other, for delivering a blow with the mallet.

Figure 21.26

Lowmann's Bone clamp which is used to hold the fracture reduction along with an implant, during plate fixation. Note the jaws and the knob with threads for obtaining a firm grip and adjustment.

Chisel

It is a sharp instrument having one edge beveled. It is used to chip the bone.

Osteotome

It is a sharp instrument having both the edges beveled. It is used to cut the bone.

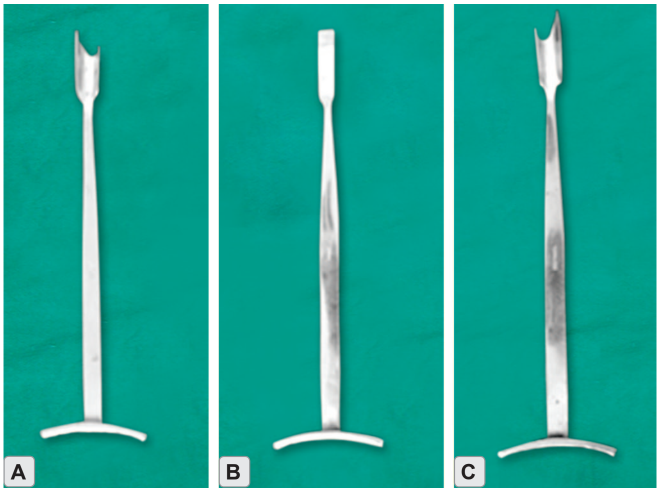
Bone Gouge

It is a sharp instrument with a trough used to gouge out the soft bone.

A mallet is necessary to drive these sharp instruments.

Figures 21.27A and B

(A) Bone cutter and (B) bone nibbler. Note that the bone cutter has sharp cutting edges at the tip whereas the nibbler has a trough with sharp cutting edges, encircling the trough, at the tip. The bone cutter is used for cutting small bone such as the phalanges, ribs, etc. The bone nibbler is used to nibble off sharp pieces of bones. The nibbled pieces get collected in the trough.



Figures 21.28A to C

(B) Smillie's meniscectomy knife. (A and C) Meiscectomy spike for the medial meniscus of left and right knee respectively. The same are interchanged (A and C are interchanged) and used for the lateral meniscus of left and right knee respectively.

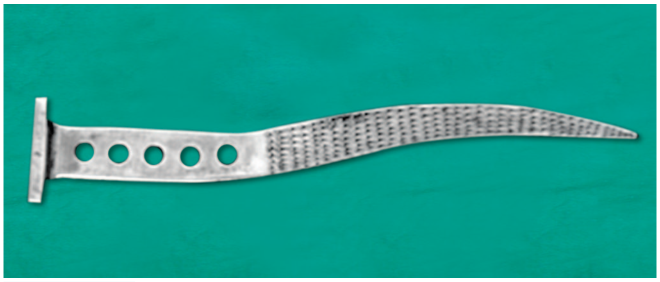


Figure 21.29

Medullary rasp which is used to rasp the medullary canal of upper 1/3 femur, for proper seating of a prosthesis in hip arthroplasty. Note the fenestrations at the upper part for insertion of a Tommy bar for extraction and a flat knob for delivering a blow for driving the rasp in.

William Arbuthnot Lane

A brilliant surgeon who was working in Guy's hospital, London, in the beginning of 20th century. He popularized what he called 'No touch technique' as a safety measure against infection by operating from a distance, using instruments with long handles. All his instruments have long handles.

William Ferguson

He was a famous surgeon of 19th century. He designed a lion toothed bone holding forceps to hold the head of the femur during disarticulation of the hip or excision of the head in tuberculosis of the hip. This forceps was also used to hold the jaw bones, the mandible and maxilla.

DESIGNS OF LIMB LENGTHENING APPARATUS

Ilizarov Fixator (Fig. 21.30)

It is a ring fixator, offering three-dimensional stability during correction.

Assembly is constructed by joining two 1/2 rings of equal diameter, chosen according to the size of the limb using ring fixation bolts and nuts (E). A minimum of four rings are to be assembled.

These rings are next connected by threaded rods (C) and the distance between the two rings is adjusted as per requirement. Next, the Ilizarov wires

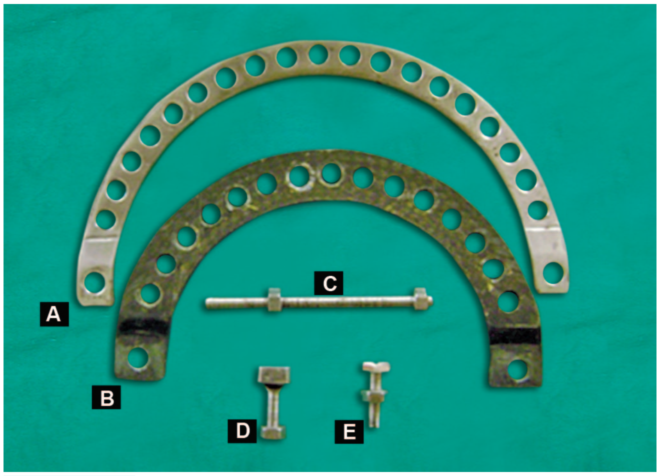


Figure 21.30

The basic components of Ilizarov ring fixator: (A) Stainless steel 1/2 ring; (B) Carbon 1/2 ring; (C) Threaded rod; (D) Wire fixation bolt and nut; (E) Ring fixation bolt and nut. Carbon rings are lighter and radiolucent when compared to stainless steel. Being radiolucent they have a distinct advantage while taking radiographs as they allow the X-rays to pass through them to give an unobstructed image of the bone whereas the steel rings obstruct the X-rays.

are passed through the bone, two at the level of each ring, preferably at right angles to each other and fixed to the ring by using wire fixation bolts and nuts (D). The wires are then tensionized by an instrument known as tensionizer. Extra-wire is cut and the cut end is bent flush with the ring. The 'Ilizarov principle' is that '***a controlled progressive distraction and/or compression leads to tissue regeneration***'. This principle is made use of and accordingly a progressive distraction or compression stress (1 mm a day) is given at the desirable site by loosening and tightening the nuts of the assembly (refer text Fig. 1.18G).

Orthofix Type (Fig. 21.31)

This is a uniplanar fixator device with an external compression-distraction (CD) unit. It has distinct advantages of ease of application and manipulation, but has disadvantage of less rigid fixation (Refer text Figs 1.13A and B) when compared to ring fixator.

Wagner Type (Fig. 21.32)

This device has a built in Turnbuckle mechanism for distraction or compression. One turn offers 1 mm movement.

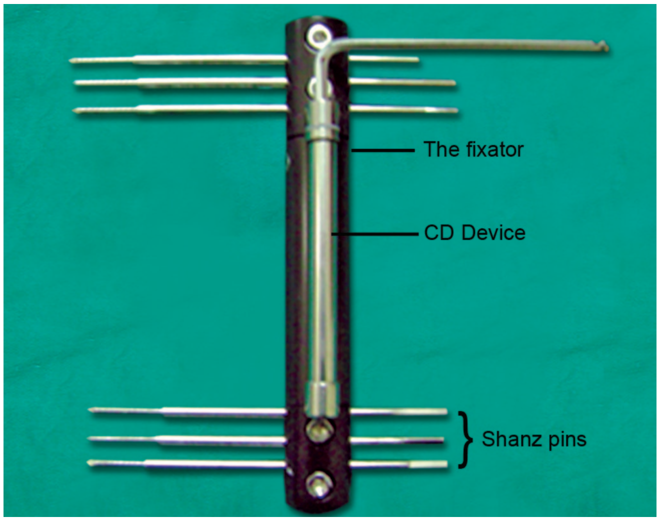


Figure 21.31

Orthofix type fixator, Uniplanar with Schanz pins *in situ* and CD* device with an Allen key inserted. One full turn of the key causes either distraction or compression by 1 mm depending on the direction of the turn given by the Allen key. *CD—Compression/Distraction device.

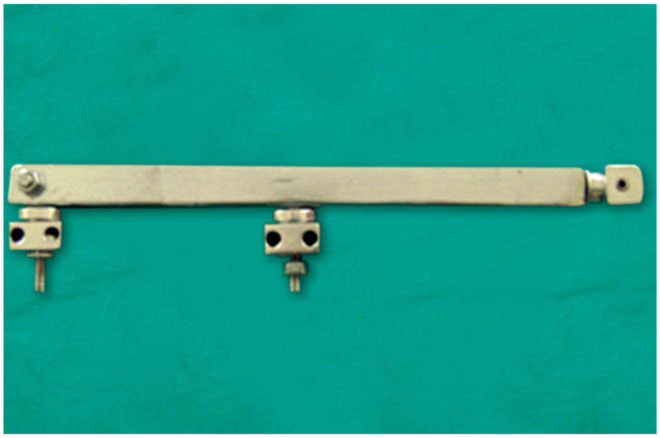


Figure 21.32

A Wagner type of lengthening apparatus. It has a fixed clamp on one side, a turnbuckle device on the other and a gliding clamp in between. This clamp glides either way according to the rotation of the turnbuckle at the other end. 1 mm translation occurs for a full turn.

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